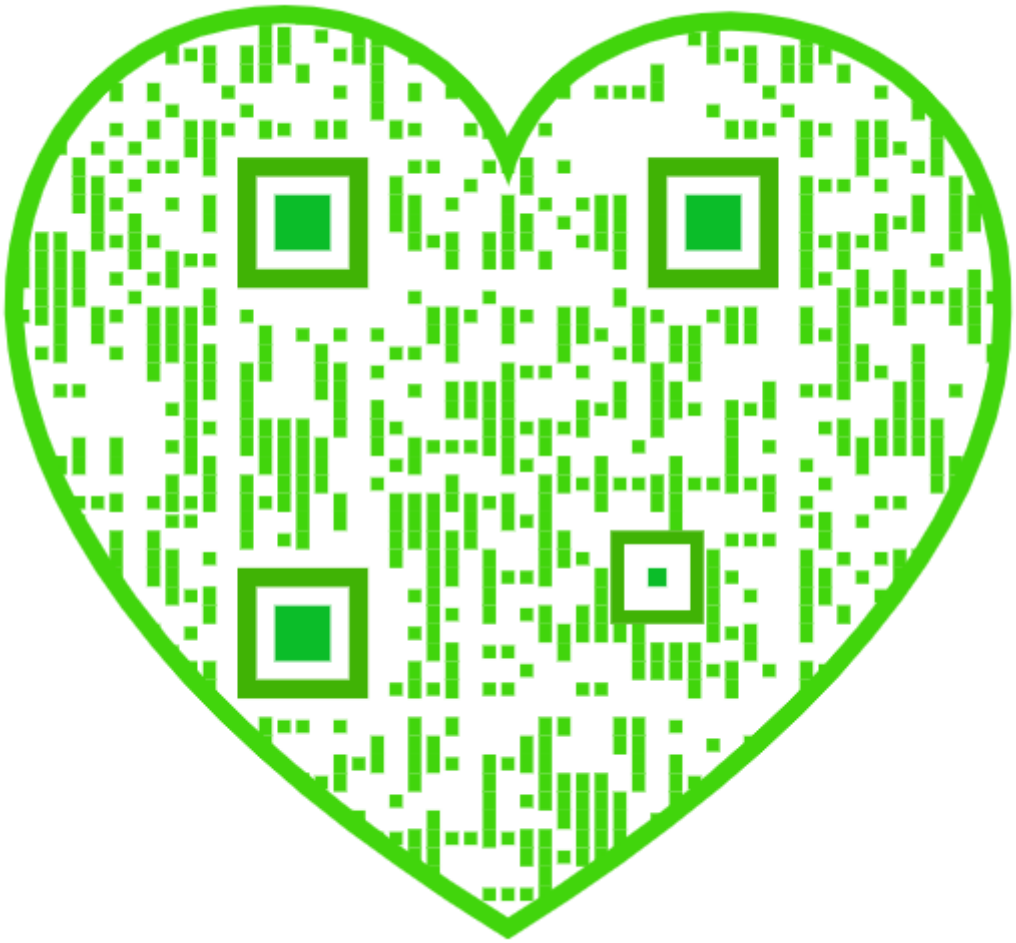

MODERN MEDICINE

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ILLUSTRATED

PHILADELPHIA AND LONDON

W. B. SAUNDERS & COMPANY

1900

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TO

OUR FRIEND AND TEACHER,

JAMES C. WILSON, A. M., M. D.,

PROFESSOR OF THE PRACTICE OF MEDICINE AND CLINICAL MEDICINE

IN

THE JEFFERSON MEDICAL COLLEGE OF PHILADELPHIA,

THIS WORK IS DEDICATED.

70726

PREFACE.

In the present era the practice of medicine includes the study of a number of specialties, such as physical diagnosis, bacteriology, the examination of the gastric contents, the urine, the blood, the feces, etc. Hence it has frequently been necessary for the student to procure separate books upon these topics. For this reason it has appeared advisable for the authors to combine in one volume, as far as possible, the essentials of these branches as applied to Clinical Medicine.

The arrangement of the special topics has been adopted to prevent repetition, to present as concise a description of allied subjects as possible, and to link more closely the various divisions. Thus, the pathogenic germ of a special disease is considered under the head of Clinical Bacteriology, rendering it unnecessary for one already acquainted with such facts to again read the morphology, the biology, the pathogenesis, etc., when dealing with the description of the disease. But if the reader is unfamiliar with the subject, such facts will be found in the section that deals with the micro-organisms that are of importance in clinical medicine.

A similar course has been followed in regard to physical diagnosis, examination of the sputum, of the stomach contents, of the blood, of the urine, and of the feces. The aim of the authors has been to give the main facts in regard to etiology, pathology, symptomatology, diagnosis, prognosis, and treatment as considered from a modern and generally accepted standpoint. The most modern medical works in English, German, and French have been freely consulted. The authors desire to express their thanks to Mr. Thomas F. Dagney, of W. B. Saunders & Co., for many courtesies extended during the progress of the work.

PHILADELPHIA, *September, 1900.*

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MODERN MEDICINE.

SYMPTOMATOLOGY AND SEMEIOLOGY.

THE science of medicine consists of a great number of specific facts, systematized and related to various sciences not necessarily pertaining to the healing art, such as biology, physiology, hygiene, etc.

Biology is the science of living things and the knowledge of vital phenomena.

Physiology pertains to the relations and functions of organized bodies, particularly the human body, to the functions of organs, and to other vital phenomena.

Hygiene is that science that treats of the laws of health.

Prophylaxis is the science that treats of prevention of disease.

Etiology has for its purpose the knowledge of the causation of disease.

Pathology treats of the causes and phenomena of disease.

Health is that condition of the living organism and of its various parts and functions that conduces to efficient and prolonged life.

"**Disease** is the perturbation of the normal activities of the living body" (Huxley).

In disease the functions alone may be deranged, such as palpitation of the heart, nausea, neuralgia; or functions and structures may both be deranged, such as in valvular disease of the heart and in croupous pneumonia; or the structure alone may be deranged, as in the formation of atheromatous ulcers, thrombi, and emboli.

Diseases are thus divided into functional and organic, although pathologically it is difficult to conceive of deranged function without some change in structure. Clinically, however, this classification must still be adhered to.

The field of medicine is divided into many branches, termed specialties, and as our knowledge of disease grows broader the limitations between the specialties become narrower; hence they are only provisional and not permanent.

The specialty with which this work is concerned is termed clinical or internal medicine. We recognize disease by two sets of phenomena, known as symptoms and signs. The organization and classification of symptoms is called **symptomatology.** The organization and classification of signs is termed **semeiology.** The *symptoms* are broadly divided as the subjective phenomena—something of which the patient complains, such as pain, nausea, vertigo.

The *signs* may be defined as objective phenomena—something which the physician must determine for himself, as the cardiac murmur, the crepitant rale, dullness on percussion.

As signs depend upon the application of the special senses by special methods employed for detecting disease during life, by anatomic changes which they produce, they are known as physical signs, and the method employed is called **physical diagnosis.**

The science of the classification of disease is termed **nosology.** Diseases may be divided into general and local. General diseases may be subdivided into infective and diathetic, etc. Local diseases relate to particular organs or tissues.

A further classification, as has previously been intimated, consists of functional and organic.

Disease may be hereditary, acquired, or congenital; specific or nonspecific; and, as regards its onset or course, acute, subacute, or chronic. Further important division relates to the character of the disease; hence a disease may be sporadic, endemic, epidemic, or pandemic.

A **sporadic disease** is one occurring singly or apart in a given locality. Examples of this form are outbreaks of cerebrospinal fever, diphtheria, etc.

An **endemic disease** is one which is always present in a certain community: as, for example, enteric fever in Philadelphia and vicinity; cholera in the delta of the Ganges.

An **epidemic disease** is one which affects a whole people or the greater number in a community. Examples of such diseases are measles, chickenpox, smallpox, etc.

A **pandemic disease** is one which affects all people in all parts of the world. Influenza is a striking example of the pandemic diseases.

EXAMINATION OF THE SURFACE OF THE BODY.

Changes in Size and Shape.—In a number of diseases the general weight of the body becomes diminished; in some few, an increase takes place. When there is wasting of the entire body, we speak of emaciation or general atrophy, but when only portions of the body are affected, the condition is spoken of as local atrophy.

In emaciation the most marked sign is wasting of the subcutaneous fat. Muscle wasting also occurs in quite a number of diseases. Local atrophy depends chiefly upon the loss of muscular tissue, but the osseous and other portions may also be involved.

Emaciation.—This can be determined by the ease with which a fold of skin can be picked up from the parts beneath. When accurate observance is necessary, the scales should be employed, and in all diseases in which this occurs, the patient should be weighed at regular intervals to determine the progress of the malady. The condition is found in the acute febrile diseases and in many chronic maladies. It takes place in enteric fever, pulmonary tuberculosis, rickets, and congenital syphilis. In children loss of weight is more rapid, but is also more quickly regained. In females great loss of weight may take place in hysteria and allied conditions. It is also a symptom of malignant disease, especially of internal organs.

Local Atrophy.—Atrophy of the skin or muscles may occur separately, or they may be jointly involved. The atrophy of the skin may result from great stretching during pregnancy, known as the lineæ albicantes. Atrophy of the hands and legs may result from lesions of the nerves or from destruction of the cells in the spinal cord. Atrophy of muscle may result from four causes: thus, atrophy from disuse; atrophy from disease of the muscles—so-called "myopathic atrophy"; atrophy from diseases of the nervous system, such as bulbar paralysis, and atrophy from disease of the joints (arthritic atrophy).

Obesity.—Obesity is a condition in which an excessive quantity of fat is present. This occurs commonly in women at the climacteric period. It is found in persons who indulge freely in malt liquors. It takes place in various forms of chronic cerebral disease and diabetes.

Dropsy is an accumulation of serous fluid in the areolar or serous cavities of the body. When the condition is general,

it is known as **anasarca**; when it is less marked or local, it is spoken of as **edema.** It increases the size of the part in which it occurs, and influences the weight in a peculiar manner. The swelling is distinguished by pitting upon pressure with the finger. This may be so great as to render the skin tense and shiny, or may be scarcely perceptible. It is most marked in the dependent parts and in the region in which there is much loose cellular tissue, as in the scrotum and eyelids. It may occur from any cause interfering with the circulation of the blood, or in conditions in which the blood is apt to deteriorate. Generally speaking, it is due to disease of the heart, kidneys, or liver. Renal dropsy shows itself often in the face and beneath the eyes, spreading downward, and may lead to anasarca. Cardiac dropsy shows itself particularly in the feet and about the ankles. Dropsy in this condition is also often due to anemia, occasionally from obstruction of the pulmonary circulation, as in emphysema. In females it may be due to varicose veins. Dropsy limited to the peritoneal cavity is spoken of as **ascites.** It is often due to obstruction of the portal circulation, occurring particularly in disease of the liver. Edema limited to the arms and upper part of the body shows mechanical obstruction in the thorax, as from mediastinal tumor pressing upon the superior vena cava. Edema limited to one leg shows obstruction of one of the veins, as in phlebitis.

Edema may be due to changes in the blood-vessel walls, as in mechanical injury of a part or in inflammation; and, lastly, from disturbance of the lymphatic system.

CONSTITUTIONAL PECULIARITIES.

The constitution of the patient depends largely upon three conditions: the osseous system, the muscles, and the adipose tissue. Depending upon the development of these three conditions, the person is spoken of as having a strong or a weak constitution.

The habitus of the patient and **cachexia** sometimes depend upon constitutional changes.

The **scrofulous cachexia** occurs in children, and the peculiar expression of the face, the lips, the prominent form of the nose, and the swelling of the lymphatic glands about the neck and lower jaw, are characteristic.

Phthisical cachexia depends upon the development of the entire body, particularly the chest. The face is thin, the eyes

sparkle, the teeth have a bluish-white appearance, and the chest shows the peculiar "expiratory form." This is also known as the phthisoid, alar, or pterygoid chest. The chest is long and narrow. The interspaces are vertical, and the costal angle is more nearly acute. The clavicles are prominent, the scapulæ stand out like wings, giving the name alar or pterygoid, and the entire chest shows marked emaciation. In this cachexia the finger-tips and the lips are often cyanotic, and the fingers show the peculiar deformity known as "clubbed finger-tips." The hair upon the head is at first well developed and thick; baldness is exceptional.

The **apoplectic constitution** shows itself in persons of small stature, with short thick necks, well-developed adipose tissue, reddened face, and in dyspnea upon slight exertion.

The **malignant cachexia** reveals great emaciation, with a peculiar pallor of the face and, usually, with normal or subnormal temperature.

THE DECUBITUS.

The position of the patient in bed shows many things that are of use in diagnosis. The normal healthy person usually assumes the dorsal position, or lies upon the side. In disease there are many changes from this position. In conditions of collapse and in many of the acute infectious diseases in which great weakness shows itself the patient is inclined to slide down to the foot of the bed and sink into a heap. This is known as the "passive dorsal"; associated with this there is often stupor and even coma. In many acute diseases of the respiratory organs involving one side, such as pleurisy with or without effusion, croupous pneumonia, and pneumothorax, the patient usually lies upon the affected side. This may be due to the fact that breathing upon the affected side is diminished in this way as the motion of the side is lessened. In pleurisy with effusion there is another advantage in lying upon the affected side, as the exudation interferes less by pressure with the sound side. In pneumonia patients not infrequently lie upon the healthy side, as pain (due to an acute pleurisy) may be caused by pressure upon the affected side. In diseases of the abdominal organs posture is often characteristic. When there is general peritonitis, the patient frequently assumes the dorsal decubitus with both legs flexed upon the abdomen. In local peritonitis, particularly appendicitis, the right leg only may be

flexed. In dyspnea and in orthopnea the patient often assumes the upright sitting posture in bed or in an easy chair, as this attitude favors the accessory muscles of respiration. In disease of the brain and spinal cord there is often retraction of the muscles of the neck, so that the head seems to sink deeply into the pillow. There may be opisthotonos, in which the body appears to rest upon the occiput and the heels; emprosthotonos, in which the body appears to bend forward; or pleurothotonos, in which the body seems bent to one side. These positions may be assumed in hysteria.

THE FACIES.

A peculiar expression of the face is often found in the dying, known as the "facies hippocratica." The facies of abdominal disease has also been described, in which the nose, chin, and cheeks stand out prominently; the eyes sink into the orbital cavity; the lips appear red or bluish-red; the expression is weak; and the entire face shows the appearance of suffering. This appearance is also seen in cholera during the stage of collapse. The facies of renal disease is quite characteristic: the eyelids are puffy, the face is swollen and edematous, and is marked by extreme anemia, showing itself in pallor.

CONSCIOUSNESS.

Disturbances of the sensorium are common in diseases grouped under the head of internal medicine. They show themselves either in an increased or a diminished psychic activity of the brain. Disturbances of the first kind are known as delirium, and those of the second variety are known as stupor or coma.

Delirium.—Delirium must be divided into **illusions,** in which the patient imagines that persons and situations are different from what he has known them to be in health, in which he mistakes one person for another, as in taking his nurse for his sister or relative, and so on; and in **hallucinations,** in which the patient imagines that he sees or hears things which in reality have no existence.

The majority of cases of delirium are due to some toxic influence. This may depend upon drugs or upon diseased conditions, such as uremia, cholemia, febrile states, and so on. Delirium is also very common in disease of the brain and in conditions of inanition. The delirium may be either low

muttering or active, and develop suicidal or homicidal tendencies.

Somnolence.—Sopor, stupor, and coma may be due to narcotic poisoning. They may occur in toxic conditions, such as uremia, diabetes, malignant disease, and conditions that produce pressure upon the brain or its membranes. It is also common in many of the acute infectious diseases.

TEMPERATURE.

The temperature of the body is taken by clinical thermometers. The bulb of the instrument may be placed in the mouth, the axilla, or the rectum. The normal temperature of the body in the mouth or axilla is 98.6° F.; in the rectum it is about a degree higher. In health there is a diurnal variation of from a degree to a degree and a half, the temperature being lower in the early morning hours (97.5° F.) and higher between five and eight o'clock in the evening (99° F.). Any elevation above 99.5° F. would constitute some morbid condition. It should, however, be remembered that serious and even fatal diseases may exist with normal or subnormal temperature.

The following classification of Wunderlich is the one usually accepted:

Range of Temperature.	*State.*	*Range of Temperature.*
Above 105.5° F.	Hyperpyrexia	Above 41° C.
Between 103°–105° F.	High fever	Between 39.5°–40.5° C.
Between 101°–103° F.	Moderate fever	Between 38.5°–39.5° C.
Between 99.5°–101° F.	Slight fever	Between 38°–38.5° C.
98.6°, or between 97.5°–99.5° F.	Normal temperature	37° C.
Between 96°–97.5° F.	Subnormal temperature	37°–36° C.
Below 96° F.	Collapse temperature	Below 36° C.

As in health, so in disease, the temperature varies between morning and evening. In the early morning there is generally a fall, called a **remission**; the rise taking place in the evening is known as **exacerbation.** It is customary in febrile cases to take temperatures every four, eight, or twelve hours. Occasionally there is a reverse in this order when the remission takes place in the evening and the exacerbation occurs in the morning. This is known as the **inverse fever** type, and appears particularly in tuberculosis.

There are three important types of fever: the *continued*, the *remittent*, and the *intermittent* types. The **continued** type is

one in which the fluctuations in the twenty-four hours are slight, not varying more than one or two degrees.

It is obvious that as the temperature is not even stable during health, it is not so in disease, and a more correct term would be the **subcontinued** type. Such a condition commonly occurs in the second week of enteric fever, the temperature being subcontinuous.

By **remittent** type is meant a fall in temperature, it not reaching the normal. This takes place in some forms of estivo-autumnal malarial fever, in tuberculosis, in the fever from impacted gall-stones, and general septic processes.

The **intermittent** type is a type in which the temperature falls to normal or below. This occurs in tertian or quartan malarial fever, in crisis from croupous pneumonia, and in relapsing fever.

The course of the fever is further divided into three stages: (1) The *onset, period of invasion,* or *initial* stage; (2) the *fastigium, acme,* or *height of the fever;* (3) the *decline* or *defervescence.*

1. **The Period of Invasion.**—The period of invasion or mode of onset of fever varies greatly. The rise may be rapid, with marked chill, or the temperature may rise slowly from degree to degree, with preceding symptoms known as *prodromes* or forerunners. An example of the rapid onset with high temperature occurs in diseases like influenza, croupous pneumonia, and malarial diseases. The form in which the fever rises gradually is illustrated by enteric fever and, occasionally, acute rheumatic fever.

2. **The Fastigium.**—This varies in different diseases, and may not in itself be a serious condition: thus, in diseases like typhus fever and croupous pneumonia the fastigium may be between 104° F. and 106° F. and remain so for several days, or only for a few hours.

In the malarial fevers the temperature, as a rule, remains high but for a few hours, whereas in enteric fever, croupous pneumonia, and typhus fever it remains at its height for a week or longer. Occasionally, pyrexia may last for several weeks or even months. This may take place in diseases like tuberculosis, syphilis, malaria, pernicious anemia, and suppurative processes.

3. The **defervescence** may be rapid or gradual, favorable or unfavorable. When the defervescence is rapid, it is known as **crisis.** When it is gradual or slow, it is known as **lysis.** In

crisis there is an abrupt fall in temperature,—three to five degrees or even more,—reaching normal or subnormal ranges, accompanied by **critical discharges,** such as profuse perspiration, copious diarrhea, or the discharge of a large amount of urine. This termination occurs frequently in croupous pneumonia, relapsing fever, and typhus fever. The fall may take place in from one to twelve or twenty-four hours. Occasionally, just before the crisis a rise takes place in the temperature. This is known as the **precritical** rise. In lysis the temperature falls gradually, taking several days to reach the normal. Discharges such as occur in crisis do not take place. This mode of termination occurs in enteric fever, bronchopneumonia, acute rheumatic fever, etc. In some diseases there may be an abrupt fall of several degrees, the temperature, however, not reaching the normal, after which the temperature rises again. This takes place in typhus fever and in croupous pneumonia, and is known as the *pseudo* or *false* crisis. It does not always follow that, as a result of crisis, the issue is necessarily favorable. The temperature may fall so low that collapse takes place, the patient dying in this condition. An unfavorable issue may take place from an ascending type, the condition being known as **hyperpyrexia.** In some diseases just before death the temperature takes a sudden upward shoot, being known as the **preagonistic** rise. The elevation may occur during the fastigium, defervescence, or in convalescence.

Occasionally, complications may arise without showing any change in the temperature-curve. Particularly is this true in acute rheumatic fever, where endocarditis and pericarditis occur without perceptible change in the fever-curve.

It is rarely possible to diagnosticate a disease from the temperature alone. Some diseases have what is known as the **typical temperature-curve,** such as the malarial diseases, enteric fever, typhus fever, relapsing fever, and croupous pneumonia, but modifications in the curve are very apt to take place, and all symptoms and signs must be taken into account before the diagnosis is reached.

The temperature in convalescence from fever is much more unstable than the normal, modifications in the curve being due to slight causes: thus, changes in diet, visits of friends, or constipation may send the temperature upward, this condition being known as a **recrudescence.** The temperature-curve is further influenced by the age of the patient, previous condition

of health, by idiosyncrasies, and by the presence of complications. In childhood the temperature is more easily disturbed than in adults, is more apt to run an erratic course, and is less subject to the ordinary laws of pyrexia. Slight ailments in children, as intestinal catarrh, may send the temperature to 103° F. or 105° F. Teething and prolonged spells of crying may have the same effect. In serious affections the temperature in childhood is liable to rise more abruptly and attain a higher range than in adults, whereas the duration is apt to be briefer. In the extremes of age, in infants and in old persons, the opposite is likely to occur. Thus, a severe illness which is ordinarily characterized by high temperature may occur with only a subfebrile fever-curve.

Alcoholics rarely show high temperature. Young adults in previous good health commonly show high temperature. As a rule, the temperature rises with the complication. An abrupt or considerable fall, if not due to crisis, is also significant of complications, especially internal hemorrhage. Occasionally, the hectic curve may occur after the crisis of croupous pneumonia. If persistent elevation of temperature takes place after convalescence has been reached, the condition is known as a **relapse,** in contradistinction to the recrudescence, in which the rise is only temporary.

Subnormal Temperatures.—A subnormal temperature occurs in quite a number of recognized morbid conditions. It takes place in diabetes, myxedema, chronic cardiac, renal, and hepatic diseases, and in many forms of mental diseases (insanity). The condition is also common in internal malignant growths.

When the temperature falls below 96° F., the condition is known as **collapse.** As has already been indicated, it may take place from crisis, from hemorrhage or perforation of the intestines, from apoplexy, cholera, or from fracture or dislocation of the spine with injury to the cord.

EXAMINATION OF THE SKIN.

A large number of diseases of internal organs are associated with some alteration in the appearance of the skin. This may be so characteristic that the diagnosis can be made alone from the examination of the skin. In diagnosis the following points must be taken into consideration : changes in color ; eruptions (exanthemata) ; changes in reference to dryness or moisture of the skin ; edema and emphysema.

The State of the Nutrition of the Skin.—In very old age the skin of the entire body seems to be thinner, in which probably the subcutaneous cellular tissues share in the atrophy. In earlier years this condition only takes place in forms of severe cachexia, in which the skin is dry and thin, with loss of tone, and when lifted in a fold, resumes its place very gradually. Many forms of atrophy of the skin have been described, but they belong to the domain of dermatology.

The color of the skin of a healthy person shows many variations, due to age, occupation, climate, and race. In some nations the pale, in others the more florid or red, complexion predominates. Any one is able to appreciate at a glance what the healthy appearance of the skin under normal conditions should be.

In disease attention must be given particularly to the following changes: (1) The pale skin; (2) the red skin; (3) the cyanotic or blue-red skin; (4) the icteroid or jaundice skin (yellow); (5) the bronze skin; (6) the gray skin.

1. **The Pale Skin.**—This may occur in physiologic conditions, especially in persons who are confined to indoor occupations and do not often come in contact with the open air. The condition is often present in disease. The pallor of the skin is noticed particularly upon those parts of the body that, under normal circumstances, due to the thinness of the skin and great degree of vascularity, are apt to show a red color, especially upon the cheeks, lips, lobule of the ear, and the under surface of the eyelids. The various shades of pale skin are divided into the alabaster color, yellowish, waxy, yellow-green, and lemon-yellow. The nutrition of the body bears no necessary relation to the pallor of the skin, as frequently persons are found with well-developed muscles and adipose tissue, characterized by extreme pallor. Duration of the pallor and particularly its cause show an influence upon the state of nutrition. Pallor of the skin under all circumstances depends upon the quantity and quality of the blood that circulates in the vessels. There may be abnormal paleness if there be either too little or impoverished blood circulating in the capillaries of the skin. The cause of the paleness may therefore be due to the decrease in the motor power of the heart or in the blood-vessels, or to a deficiency in the amount of hemoglobin. The diagnosis between these two conditions can, however, only be made with certainty by an examination of the blood. Primary pallor may be due to syncope or fainting, to

emotion, fright, to cold, as in the chill of fever, in spasm of the capillary vessels, and in allied conditions.

Paleness is encountered in persons suffering from fatty degeneration of the heart. This may be due to the diminished amount of force of the heart muscle, in which condition the vessels of the skin are not properly filled. Pallor furthermore takes place after hemorrhages, whether they be large or small and frequently repeated. It occurs in bleeding from the nose (*epistaxis*), bleeding from the gums, spitting of blood (*hemoptysis*), vomiting of blood (*hematemesis*), blood from the intestines (*enterorrhagia*), blood in the urine (*hematuria*). The condition is also found in internal hemorrhages, such as bleeding into a pulmonary cavity, pleura, and pericardium, from malignant disease, tubercle, or scurvy. It may occur from parasites, especially those appearing in the bowel, such as the anchylostoma duodenale. Pallor of the skin due to blood loss may be also, although indirectly, associated with disease of the kidney, purulent exudation in the pleura, pericardial or peritoneal sacs, or due to accumulation of pus in other localities. Persons with chronic disease of the gastro-intestinal tract in which digestion is interfered with are apt to suffer from pallor. Ulcer of the stomach and chronic catarrh of the stomach and bowel will cause pallor. It is likely to affect persons suffering from intestinal parasites, particularly the various varieties of tapeworm. There is pallor in diseases of the blood. It is a symptom of chlorosis, leukemia, pseudoleukemia (Hodgkin's disease), and pernicious anemia. Pallor of the skin is further found in association with acute and chronic infectious diseases, such as malaria, acute rheumatic fever, diphtheria, tuberculosis, and malignant disease. Lastly, it may be due to metallic poisoning, particularly lead, mercury, and arsenic.

2. The Red Skin.—The red appearance of the skin may also be noticed best upon the lips, cheeks, mucous membrane of the conjunctiva, and the lobule of the ear. Persons who expose the face to the open air are likely to have a red skin. It is noticed especially in those who labor in the sun. It may be temporary, appearing and disappearing rapidly. This may occur in nervous, excitable persons, or under slight psychic impressions, as in blushing. Persons who are continually exposed to heat often have red skin, especially cooks, smiths, and so on. The red skin is common after warm bathing. It may depend upon the active dilatation of the vessels of the skin, or

to an increase in the total mass of the blood in the vessels of the skin, or to an increased amount in the corpuscles and coloring-matter of the blood. Abnormal redness of the skin may occur in hemicrania. It is present most frequently in fevers. It is often noted in plethoric conditions, in individuals called full-blooded, and in poisoning from drugs such as atropin.

3. The Blue-red or Cyanotic Skin.—This shows itself most prominently upon the parts that are normally bright red, such as the lips, cheeks, mucous membranes, and the finger-nails. If the condition is marked, it may show itself all over the surface of the body. Occasionally, cyanotic areas are combined with pale skin, when the condition is spoken of as **livid.** Cyanosis is due to the accumulation of carbonic acid in the blood and a deficient amount of oxygen, the blood having a venous or hypervenous character. It may arise from an interference of the change of gases in the lungs, preventing the blood from accumulating sufficient oxygen and not allowing itself to lose sufficient CO_2; or it may be due to a diminution in rapidity of the circulation of the blood in the capillaries, so that it gives off more oxygen and absorbs less CO_2, hence the condition arises particularly in disturbances of the respiration or circulation. Should both conditions be coexistent, a high grade of cyanosis will develop.

Diseases of the Respiratory Organs Causing Cyanosis.—The condition may be caused by a narrowing of the large air-passages, so that the atmospheric air can only partly gain access to the alveolar structure of the lungs. Acute and chronic inflammations of the pharynx or larynx, retropharyngeal abscesses, or diphtheria may lessen the lumen of the trachea and produce cyanosis. It may also result from croupous bronchitis, bronchial asthma, paralysis of the vocal cords, especially of the dilator of the glottis (crico-arytenoideus posterior), emphysema, and disease of the alveolar tissue of the lungs, or of the parenchyma of the lungs, or a combination of these conditions. It may occur from the presence in the air-passages of foreign bodies, tumors, goiters, aneurysms, or from enlarged glands. It is caused by all forms of consolidation. Tuberculosis may produce a high grade of cyanosis. Compression of the lung from pleural and pericardial effusion or accumulation of gases in the serous sacs may produce extreme cyanosis. It rarely results from pressure upward from the abdominal organs, such as meteorism, tumors, and large accumulation of fluid in the peritoneal sac. It may result from paralysis of

the respiratory muscles, bulbar paralysis, peripheral neuritis, paralysis of the diaphragm, spasm of the respiratory muscles, epilepsy, trichinosis, occasionally from hysteria, and from various diseases of the muscular tissues themselves, such as the myopathic form of progressive muscular atrophy and myositis ossificans.

Cyanosis due to disease of the circulatory apparatus may be due to lesions of the heart itself or of the peripheral vessels. Contraction of the smaller vessels at the periphery, in slowing the circulation, may produce cyanosis, as from the influence of cold. Peripheral cyanosis may be due to the interference of the return of the blood supply, as in preparing a part for bleeding. The same condition takes place in thrombosis of the veins or from compression of tumors upon a part. Causes due to the heart itself may be found in disease of the valves of the heart, in failure of compensation from valvular disease, cyanosis often developing to a high degree. Cyanosis is pronounced in stenosis of the pulmonary valve, and is a prominent symptom in congenital valvular disease. Disease of the heart muscle itself, whether it be due to disturbance of innervation, inflammatory or degenerative processes of the muscle substance itself, produces cyanosis, as this diminishes the force and activity of the circulatory apparatus. A similar result may occur from effusions or accumulation of gas in the pericardium. Cyanosis of a high grade is often encountered from the toxic effects of drugs, such as nitrobenzol, from the coal-tar products, such as antifebrin, antipyrin, phenacetin, kairin, thallin, and others. It may occur from the inhalation of illuminating gas. The cyanosis in such instances is due to the fact that the blood loses its power of absorbing sufficient oxygen.

4. The Icteroid or Jaundice Skin.—The icteroid skin develops when bile or bile pigments circulate in the blood stream. The condition is easily recognizable. In mild grades there is a light sulphur-yellow appearance of the skin. This is always most pronounced in the conjunctiva. It can easily be discovered, if present to a slight degree, by taking a glass slide and pressing it upon the mucous membranes of the tongue, when the yellowish color will be noticed. If the condition is more intense, the lemon-yellow will develop into a tinge of darker yellow—even to a brownish-yellow color known as *melasicterus*. Jaundice can not be detected by artificial light, since yellow artificial light does

not enable one to distinguish between white and yellow. In mild cases it is best detected in the conjunctiva. Occasionally, in the negro the conjunctiva shows a yellowish cast. This does not necessarily indicate jaundice. As jaundice begins to develop it does not show itself in all parts of the skin with equal intensity: the yellowish appearance is most pronounced in parts in which the epidermis is thin, such as the angles of the mouth and the alæ of the nose, the forehead, and the skin of the neck, the chest, abdomen, and extremities being the last parts to be affected. Jaundice is usually more intense at the flexures of the joints. At the commencement of the icteroid appearance the yellow color is particularly due to the blood plasma that has accumulated bile pigment and so produced the abnormal color; later, the cells of the rete Malpighii absorb bile pigment and retain it for some time. This explains the slow disappearance of jaundice and the return to normal color in many diseases in which jaundice has persisted for any length of time. Jaundice is divided into hepatogenous and hemohepatogenous jaundice.

Hepatogenous Jaundice.—Hepatogenous jaundice is the jaundice of stagnation, being the result of an interference with the flow of bile from the liver or the bile-ducts to the common bile-duct into the intestine. This produces stasis. The bile becomes thin, being absorbed into the blood. This results from catarrh of the bowel, especially the duodenum, affecting the common bile-duct, tumors pressing upon the duodenal opening of the common duct, cancer of the head of the pancreas, parasites in the common bile-duct, and gall-stones lodging there. The hepatic duct may also be compressed by tumors or cysts (*cancer, echinococcus*), or obstruction in the small bile-ducts of the liver. Compression of branches of veins of the liver or catarrh of the small bile-duct possibly produces jaundice. The symptoms produced by jaundice are, first, the yellow skin, particularly in the parts that have just been enumerated; the appearance of bile pigment in the urine; clay-colored stools; slowing of the pulse; headache, and other nervous manifestations. There may sometimes be subnormal temperature, and, if the condition continues, stupor and coma with convulsions may develop. Such a condition is known as *cholemia*.

Hemohepatogenous Jaundice.—For some time it has been well established that jaundice occurs in certain forms of poisoning and in the infectious diseases. In such instances the icterus is due to the marked alteration in the blood, and is character-

ized chiefly by an alteration in the erythrocytes and in the hemoglobin. This form of jaundice takes place in poisoning by chloroform, phosphorus, ether, chloral, potassium chlorate, and arsenic. In the infectious diseases it is most noticeable in yellow fever, pyemia, and, occasionally, in croupous pneumonia. In either instance, however, it must be remembered that the liver itself may bear some share in the production of the icteroid condition. In the hemohepatogenous variety the urine does not, as a rule, show bile pigments, nor are the feces likely to be clay colored. Only in grave alterations of the hemoglobin and corpuscles does hemohepatogenous jaundice show itself.

Icterus Neonatorum.—This is the jaundice of the newborn, and may be either benign or malignant. The explanation of the condition is very doubtful. It has been attributed to purely mechanical conditions, such as the sudden decrease of pressure in the portal veins, also alterations in the blood itself. Neither of these explanations is satisfactory.

5. The Bronze Skin.—In 1855 Addison called attention to the peculiar bronze-like discoloration of the skin that was almost constantly associated with the chronic diseases of the suprarenal capsules. The bronze skin shows itself by a brown-grayish to black discoloration, especially upon the exposed parts of the body, such as the face and hands. From these parts it may gradually extend all over the body, excepting the nails and cornea, which usually remain clear. The mucous membranes are similarly affected, the lips, however, showing the discoloration less. It is due to the deposit of pigment in the rete Malpighii. Pressure with the finger does not cause the discoloration to disappear. The administration for a long time of some drugs, particularly arsenic, may cause a similar discoloration of the skin and mucous membranes, which does not always disappear even after the arsenic has been discontinued.

6. The Gray Skin.—After the salts of silver have been administered for some time, fine particles begin to deposit in the skin, sweat-glands, and even in some of the internal organs, as in the kidneys or intestines. The color may be grayish or blackish, and especially affects the face and hands. In well-marked cases the mucous membrane of the mouth may also show discoloration. This condition is known as *argyria*.

ERUPTIONS (EXANTHEMATA).

There are certain eruptions that have a general diagnostic value, such as *herpes*, *roseola*, and *sudamina* (*miliaria*).

Sudamina occur in all diseases in which sweating is a prominent symptom. They show themselves by a slight elevation of clear fluid under the skin, being about the size of an ordinary pinhead. The place at which this eruption most commonly appears is upon the lower part of the abdomen. It occasionally occurs in health, owing to too prolonged bodily exertion or as a consequence of exposure to the direct rays of the sun. They appear commonly during the course of many acute infectious diseases. They are also found in chronic pulmonary tuberculosis, in miliary tuberculosis, in acute rheumatic fever, in enteric fever, especially toward the end of the third week, and in pyemia. Sudamina may also often show themselves in the stage of sweating that takes place just prior to death.

Herpes also show themselves by the development of small vesicles. These are most often larger than those of sudamina, and they occur in groups. They are occasionally confluent. The vesicles at first contain clear fluid, which later may become cloudy and occasionally yellowish, for pus-formation takes place. They soon desiccate, leaving a crust that does not form a cicatrix upon the skin. The most common site for the appearance of the herpes is in the neighborhood of the mouth, especially upon the lips, when the condition is known as herpes labialis; or it may appear at the nose, when it is known as herpes nasalis; or near the eyelids, when the condition is known as herpes palpebralis. The diseases most commonly found associated with herpes are croupous pneumonia, cerebrospinal fever, malaria, relapsing fever, and erysipelas. The eruption is occasionally noted in gastro-intestinal catarrh. It sometimes appears during the course of menstruation, and may depend upon nervous influences, such as fright, anxiety, etc. The eruption is exceedingly rare in enteric fever and in typhus fever, and is said never to occur in tubercular meningitis.

The **roseolar eruptions** are of especial value in some of the most important diseases. They show themselves by round, rose-red, slightly raised points (maculopapular) that disappear upon pressure. They are usually found scattered upon the lower part of the abdomen, the chest, the back, particularly between the shoulder-blades, and rarely upon the extremities and the face. They are from two to three lines in diameter.

The eruption very closely resembles flea-bites. This is the characteristic (specific) eruption of enteric fever. It occurs about the end of the first or the beginning of the second week. The individual eruption lasts from two to three days, and appears and disappears in successive crops. The eruption of measles may occasionally resemble this, but in morbilli the eruption is coarser, more raised, does not disappear upon pressure, and is found prominently upon the face. Eruptions may appear from the use of certain drugs or poisons, and may show themselves in various forms. They may occasionally resemble the acute exanthematous diseases, such as scarlet fever and measles, and may give rise to difficulty in diagnosis. The drugs that are most likely to cause eruptions are quinin, antipyrin, salicylic acid, morphin, atropin, strychnin, various balsams, particularly copaiba, iodin, bromin, and substances that are applied locally, such as turpentine and mustard.

When **hemorrhages** occur under the skin, the condition is known as *purpura*. When it occurs in small spots, they are known as *petechiæ*. When the eruption appears in large patches, it is known as *ecchymosis*.

Cicatrices.—Cicatrices upon the skin are often of importance. The eruption of smallpox, especially upon the exposed parts,—the face and the hands,—leaves a well-marked cicatrix. Many of the syphilitic eruptions leave prominent cicatrices. The striæ, or the scars of pregnancy, show themselves upon the lower part of the abdomen and upon the thigh. Similar scars sometimes occur after marked edema of the abdomen. The scars from injuries have no importance in clinical medicine.

EDEMA.

This has already been partially described in a previous section; further reference must, however, be made at this point. By edema is meant an abnormal accumulation of fluid in the tissues: accumulation particularly in the cellular meshes and lymphatic spaces, being a transudate from the blood-vessels. The fluid may be absorbed by the lymphatics, and again find entrance into the blood. The condition is recognized by an increase in volume in the affected part and the swelling filling up depressions and cavities, the skin appearing puffy and pitting upon pressure. The skin is usually slightly shining and smooth, often accompanied by pallor, due to the diminished circulation. (See p. 20.)

EMPHYSEMA OF THE SKIN.

By this is meant entrance of air into the cellular tissues, and, depending upon its distribution, it may be spoken of as circumscribed or local and general or diffused. The recognition of the condition is not difficult. The affected parts are raised, and upon palpation give a peculiar crepitant sensation to the finger similar to that which is encountered when normal lung tissue is felt. Upon pressure with the finger a slight temporary depression may occur, and the condition may resemble edema, but the resemblance is only slight, for in edema the sensation of crepitation is entirely absent. Emphysema may show itself in two forms: first, the so-called spontaneous emphysema of the skin, which may depend upon inflammations or abscesses or large extravasations of blood without having communication with the external air, in which gas is developed in the subcutaneous tissues. It may be due to the gas-producing bacilli (bacillus aerogenes capsulatus), and belongs to the domain of surgery. The second form is known as aspiration emphysema, and may be due to the entrance of air from an external wound, such as from tracheotomy. Many of these causes also belong to the domain of surgery, internal medicine concerning itself only with those cases in which air enters from diseases of internal organs. Such conditions may arise from ulcerative processes of the trachea and bronchi, in which perforation may take place, in disease of the pulmonary structure, such as cavities in the lungs, which may ulcerate into the chest-wall and develop a communication with the subcutaneous tissue. Pulmonary alveoli may rupture from great intrathoracic pressure, such as severe cough. This may take place particularly in children with whooping-cough, bronchitis, or emphysema, from sharp crying, severe exertion, such as blowing upon wind-instruments, glass-blowing, etc. Air may enter from under the pleura into the interalveolar tissue, reach the mediastinum, and thus get into the subcutaneous tissues of the neck and spread onward; finally, emphysema of the skin may occur from disease of the esophagus, stomach, or intestines. This may result from rupture, from ulceration, malignant disease, or foreign bodies. It is a prominent occurrence in perforation of the bowel or stomach in which the abdomen soon becomes filled with gas, and is a symptom of peritonitis due to perforation.

MOISTURE OF THE SKIN.

In health the secretion of sweat is variable, and this condition is apt to be exaggerated in disease. When sweating occur to a high degree, the condition is known as *hyperhidrosis* When sweating is much diminished, it is called *hyphidrosis* and when sweating is arrested altogether, the skin being perfectly dry, it has received the name of *anidrosis*. Marked sweating over the whole surface of the body is called *hyperhidrosis universalis;* if it be confined to a part, *hyperhidrosis localis* is spoken of. If it appear only upon one side of the body, it is spoken of as *hemidrosis*. Many influences may bring about these conditions, such as changes in the blood and lymph. The accumulation of urinary products, of carbonic acid, the auto-infections, bacteria, chemic bodies, and poisons may suppress or produce perspiration. They may act either upon the sweat-glands themselves or directly upon the nervous system. Sweating occurs during convulsions due to the increased amount of muscular work and increased activity of the heart. On the other hand, it may be entirely absent in epileptics and in hysteria. It results from great excitement, fright, severe pain, after warm bathing, occasionally from the influence of atmospheric heat, and from certain drugs, as sudorifics (pilocarpin). Dyspnea may be accompanied by sweating. Sweating occurs in many of the febrile diseases, especially in diseases ending by crisis, such as croupous pneumonia, relapsing fever, or malaria. It takes place in other febrile conditions that are marked by fall of temperature, such as pyemia, the night-sweats of phthisis, the latter stages of enteric fever, and in the cold sweats of collapse. Acid sweats are met with particularly in acute rheumatic fever. Local sweating is often a prominent symptom in organic disease of the nervous system. It occurs in migraine, localized disease of the brain, pressure upon the sympathetic nerve, and is often a symptom in exophthalmic goiter and in many mental diseases. Diminished sweating or complete anidrosis occurs in diseases characterized by continued high temperature. It may be due to the loss of considerable amounts of fluid from the tissues, as in diseases of the bowel, as severe diarrhea, or to contracted kidney and diabetes.

THE PULSE.

From contraction of the ventricles, a certain amount of blood is sent into the aorta and thence into the peripheral arteries. In this way the lumina of the arteries are widened, which shows itself in two directions—in an increase of their transverse diameters and in an extension of their long diameters. This gives rise to pulsations that correspond very closely to the systolic or first sound of the heart. For purposes of examination the right pulse is chosen, on account of its convenience. The pulse depends principally upon three conditions: the amount of work done by the heart, the quantity of blood in the artery, and the condition of the arterial wall itself. In a great many diseases the pulse is of importance in diagnosis. For purposes of examination palpation of the pulse or feeling of the pulse (sphygmopalpation) is all that is ordinarily required. For purposes of exact examination an instrument is used, by which means pulse-tracings are obtained. This instrument is called a sphygmograph, and the tracing is called the sphygmogram. An instrument has been invented for measuring the pressure of the pulse, known as a sphygmomanometer.

Palpation of the Pulse.—Sphygmopalpation.—For purposes of examining the pulse the second or third finger of the right hand should be placed upon the radial artery, at or near the styloradial process. Firm pressure should be avoided, as this may produce changes in the rhythm and frequency. Three things must be noticed in examining the pulse: *its frequency*, *its rhythm*, and *its quality*.

Pulse Frequency.—In the ordinary healthy adult the pulse varies between sixty and eighty beats a minute: seventy may be taken as the average pulse. For purposes of convenience the pulse is counted with the second-hand of the watch, and for exact study an entire minute should be counted. Beginners should not content themselves with counting for fifteen seconds, as many circumstances may produce errors. Frequently, when the pulse is first felt it is apt to be slightly more rapid than it would be at the latter part of the minute, on account of excitement due to the examination. The pulse frequency normally depends upon the age and position of the patient, the taking of food, excitement, exercise, respiration, the temperature of the air, and the influences of certain poisons. In the first few weeks following birth the pulse is highest, and it

gradually diminishes up to the twenty-fifth year of life. It remains at its minimum between the ages of twenty-five and fifty, and increases again as old age is reached. During fetal life the pulse is from 135 to 140 a minute. During the first year of life it averages about 134 a minute; from the first to the second year, about 110 a minute; from the fifth to the sixth year, about 98 a minute; from the tenth to the eleventh year, about 88 a minute; from the fifteenth to the twentieth year, 72 a minute; from the twenty-fifth to the fiftieth year, 70 a minute; from the sixtieth to the eightieth year, between 74 and 79 a minute. These are the observations of Eichhorst. The influences of sex depend upon the fact that the pulse is more frequent in females than in males. This pulse frequency even exists during fetal life, and led Frankhäuser to attempt to foretell sex in the fact that the pulse of the female is from five to ten beats a minute more frequent than that of males. This difference is less marked between the ages of twenty-five and thirty, when the pulses are more uniform. Position of the person is of importance. It shows its influence in the fact that the pulse is slowest in the recumbent posture, and most frequent in the erect posture. The time of day is also not without importance. The pulse-beats are more frequent in the early morning hours, between three and six o'clock, and the pulse reaches its maximum about eleven o'clock in the morning; the frequency then declines until about two o'clock in the afternoon, when it begins to rise, and continues until between six and eight o'clock in the evening, when it reaches its second maximum; it then begins to decline in frequency until midnight.

The *taking of food* has a decided influence upon the pulse frequency. Thus, after meals, especially if hot, indigestible food and liquors are partaken of, there is a marked increase in the frequency of the pulse. Abstinence from food or fasting decidedly diminishes the pulse-rate. Exercise increases the pulse-rate. Deep respiration has a similar effect. The temperature of the air has an influence; thus, a high temperature increases the frequency of the pulse, and low temperature slows the pulse. Atmospheric pressure, as in a pneumatic cabinet, decreases the pulse-rate. Rarefied air, as in mountain regions, on the contrary, increases the pulse-rate. Influences upon the pneumogastric nerve produce variations in the pulse; thus, irritation decreases, whereas division or paralysis of the nerves increases, the pulse-rate. Drugs have an important in-

fluence; thus, digitalis and calabar bean diminish the rapidity of the pulse; veratrum and nicotin in small doses slow the pulse; in larger doses they increase the pulse. Atropin increases the pulse-rate.

Morbid conditions show their influence upon the frequency of the pulse in two ways: they may slow the pulse (*pulsus rarus*) or they may increase the pulse-rate (*pulsus frequens*). Slowing of the pulse is present in the following conditions: in jaundice the pulse-rate is frequently, although not invariably, diminished. It may fall to fifty or forty or even lower a minute. It has lately been proved by Legg and others that bile pigment absorbed in the blood has an influence upon the heart muscle and ganglia, and in this way a slowing pulse-rate is produced. Degeneration of the heart muscle produces a slowing of the pulse. Stokes called attention to the fact that in fatty heart and in arteriosclerosis of the coronary arteries, also in myocardiac affections, the pulse frequency is decreased. A case has been recorded in which, from this cause, the pulse-rate fell to eight a minute. In stenosis of the aortic valves Traube has shown that in the majority of cases the pulse-rate is slowed as the coronary arteries and the heart muscle do not receive sufficient blood. In disease of the central nervous system the pulse is often slowed in a remarkable degree. From increased pressure upon the cerebral substance from growths, from hemorrhage, from hydrocephalic fluid in the course of basilar meningitis, or from inflammation of other parts of the vagus (pneumogastric nerve) the pulse frequency is often markedly diminished. In suddenly lowered blood pressure in the arterial stream from large hemorrhages or from bleeding, as a therapeutic measure, the pulse frequency is decreased. The same effect may be observed after aspiration of a pleural or pericardial effusion. In the crisis from acute infectious diseases the pulse-rate is frequently diminished. In conditions of inanition either due to want of food or stenosis of the esophagus, or in chronic catarrh of the stomach and bowel, the pulse-rate may be lessened. Occasionally, it occurs in the course of acute rheumatic fever without intercurrent cardiac affection and in parturition; this Olshausen attributes to lipemia. In some of the intoxications—thus, from lead or alcohol—the pulse-rate may be decreased.

Increase in the Pulse-rate (Pulsus Frequens).—Increase in the pulse-rate occurs under the following pathologic circumstances: In fever it is one of the most constant symptoms; in

this condition one should always suspect complications if, during the course of fever, the pulse-rate should diminish in frequency. In the majority of cases the pulse frequency at the height of the fever shows a certain proportion. According to Liebermeister the pulse-rate rises to eight beats a minute for each degree in the rise of the fever; however, many exceptions occur to this rule, and many factors may influence the condition. The prognosis in all febrile diseases should be considered extremely serious if the pulse-rate should rise to 160 a minute or over. When febrile diseases occur in weak or cachectic individuals, the proportion between pulse and fever is apt to be higher; thus, in febrile conditions in such individuals the pulse is apt to be very much higher than the temperature would indicate. The same is true in febrile diseases occurring in infancy, and in febrile diseases taking place in persons affected with valvular disease of the heart. In enteric fever and in yellow fever a lower pulse-rate is usually encountered than in other febrile diseases. In acute miliary tuberculosis, septicemia, and pyemia, the pulse-rate is very high.

In collapse if the temperature should fall to 96° F. or below, the pulse-rate is very apt to be greatly increased in frequency. The prognosis in such conditions is always serious. The pulse-rate may become extremely rapid, amounting to 200 a minute or more. In such cases it is almost impossible to count the pulse-rate with accuracy. It should be done by counting the contraction of the ventricles by auscultating the heart, or every other pulse-beat may be counted and the number doubled a minute. In paralysis of the pneumogastric nerve, which may be due to disease of the central nerves at the origin of the vagus or any part of its course, the pulse-rate is markedly increased. Paralysis of the pneumogastric occurs most frequently from pressure from enlarged lymphatic glands, such as might result during the course of pseudo-leukemia (Hodgkin's disease). In certain neuroses of the heart muscle, such as nervous palpitation, stenocardia, and exophthalmic goiter (Graves' disease), a marked increase in the pulse-rate may take place. In many valvular diseases of the heart, especially during the stage of ruptured compensation; in cases in which there is great difficulty in the flow of the blood through the arterial system, such as from the accumulation of pleuritic exudation and also from the accumulation of fluid in the pleural sac pressing upon the large vessels, the

pulse frequency is apt to be increased. In diseases of the lungs in which there is difficulty of the blood emptying itself from the pulmonary artery the pulse-rate is more rapid. Pain markedly increases the rapidity of the pulse. In diseases of the blood, such as chlorosis and pernicious anemia, the pulse-rate is often increased. A very slow pulse—under 60 a minute—is known as *bradycardia.* A pulse-rate of 120 or over is known as *tachycardia.*

Rhythm of the Pulse.—Three varieties of rhythm of the pulse have been described: the *rhythmic*, the *allorrhythmic*, and the *arhythmic* (irregular) pulse. In normal healthy individuals the pulse shows regular rhythmic intermission. This is known as the *rhythmic* pulse. An experienced finger can readily detect this condition, and can easily detect alterations in the normal pulse. A double pulse-beat is known as *dicrotism.* This condition takes place in fevers (enteric fever), convalescence, and anemic conditions. Frequently there is a decided pause between the first strong beat and the second weaker beat; occasionally it may happen that there may be more than one beat following the first. In rarer instances the first beat may be the weaker one, and the second beat the more powerful. The latter condition has been called the *hyperdicrotic pulse.* The term allorrhythmia was introduced by Sommerbrodt. By this term it is understood that although the pulse does not conform to the normal rhythm, it nevertheless shows a decided periodicity of rhythm. To this variety belong the *pulsus paradoxus*, *pulsus bigeminus*, and the *pulsus alternans.* Allorrhythmia must be very pronounced to be appreciated by the finger, and experienced and practised observers may err unless sphygmographic tracings are taken of such pulses. The *pulsus paradoxus* is one that becomes smaller and may even entirely disappear during inspiration. The *pulsus bigeminus* has for its characteristic that it is made up of two beats, one following the other, succeeded by a somewhat prolonged pause. The *pulsus alternans* shows a regular changeability between a large and a small pulse. By the *arhythmic pulse* is meant one that shows decided irregularity in reference to the succession of beats. It has sometimes been called the *pulsus intermittens.*

Quality of the Pulse.—Three things must be noted in reference to the quality of the pulse: *expansion*, *strength*, and *size.* In reference to the expansion, a rapid pulse (*pulsus celer*) and a slow expansion pulse (*pulsus tardus*) may be recognized.

The *pulsus celer* shows itself in the fact that the artery reaches its maximum of expansion in a very short period of time. This is apparent to the finger by the fact that the beat rapidly disappears. In the *pulsus tardus* the opposite condition is noted. Both these pulses occur commonly in diseases of the aortic valves, aortic insufficiency exhibiting the pulsus celer, and stenosis of the aortic valves showing the pulsus tardus. In very many cases the pulsus celer is also the frequent (rapid) pulse. An important variety of the pulsus celer, as has already been indicated, occurs in aortic regurgitation. It is known as the *trip-hammer, water-hammer, receding*, or *Corrigan pulse*. The pulse is more characteristic when the ventricular contractions are short, and the less the flow of the blood is hindered from the small vessels and veins, the more rapidly the artery contracts. It is, therefore, easy to understand how the pulsus tardus occurs in pulmonary emphysema, arteriosclerosis, lead colic, and in a great variety of diseases characterized by severe pain. In reference to the strength of the pulse, a hard pulse (*pulsus durus*) and a soft pulse (*pulsus mollis*) are spoken of. The strength of the pulse is diagnostic from the degree of pressure required to prevent the pulse from expanding. The strength of the heart and the tension of the arterial wall are factors in the production of a hard or a soft pulse. The beginner must be careful not to confound the hard pulse with arterial sclerosis. In the latter condition the pulse feels like a whip-cord under the fingers, which is not the case in the *pulsus durus*. The hard pulse is encountered in hypertrophy of the left ventricle; hence it is found in aortic insufficiency and in contracted kidney, in attacks of lead colic and in inflammatory conditions, especially in those characterized by pain, such as peritonitis. In reference to the size of the pulse, we speak of the regular pulse (*pulsus equalis*) and the irregular pulse (*pulsus inequalis*); a full and an empty pulse (*pulsus plenus* and *pulsus vacuus*); a large and a small pulse (*pulsus magnus* and *pulsus parvus*).

The Regular and the Irregular Pulse.—In the irregular pulse some pulse-waves may be larger than others. Very often there is irregularity as to frequency and size. A flat and empty pulse may depend upon the walls and the size of the lumen of the artery. A large pulse usually occurs in healthy individuals during the middle periods of life. The pulse is smaller in childhood and in old age. Men have a larger pulse than women, and the pulse is larger after meals than

before. A large pulse is apt to be slower than a small pulse. If anatomic relations of both sides of the body are symmetric, the radial pulse will be of even volume and rhythm. If abnormalities of the arteries exist, the pulse is apt to show variation as to time and quality (comparing the two sides). The symmetry of the pulse is also influenced by aneurysms, such as of the innominate artery, the subclavian, axillary, and the brachial. Pressure upon certain arteries may give the same variation—from tumors, large pleural effusions, pneumothorax, dislocation and fracture, enlarged glands, thrombosis, embolism, and tumors of the vessel-wall.

SPHYGMOGRAPHY OF THE RADIAL PULSE.

The idea of producing the tracings of the pulse originated with Vierordt. The instrument used for this purpose is known as the sphygmograph. The two instruments that are most frequently employed are the Marey and the Dudgeon

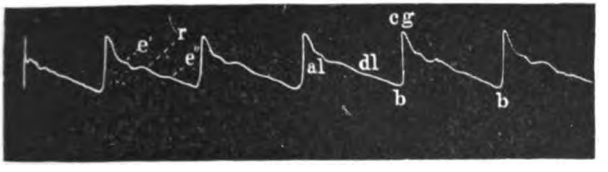

Fig. 1.—Normal pulse-curve in a healthy man aged twenty-five years (after Eichhorst).

sphygmographs. Sphygmographic tracing records the pressure of the pulse in the artery, but it must be understood that the measurement is only relative and that an absolute measure of the size of the pulse or of the internal pressure of the artery can not be obtained in this way, as the height of the pulse-wave varies with the position of the instrument with reference to the artery and the position of the pad that receives the tracing of the pulse. It follows from this that very little dependence should be placed on the height of the pulse-wave, and only the forms should be observed.

In health the pulse-curve, which is obtained by means of the sphygmograph, shows ascending and descending lines, elevations, and depressions corresponding with the collapse and expansion of the artery. The apex-curve (*cg*) and curve

at the base (*b*), as can be seen in figure 1, need no explanation. The line *al* is known as the *ascension line*, which is almost perpendicular. The rise follows quickly. The descent is more gradual and may show several small waves, which usually may be differentiated as a decided elevation (*r*), a backward stroke elevation or recoil due to the closure of the semilunar valve, and two, occasionally three, or only one,

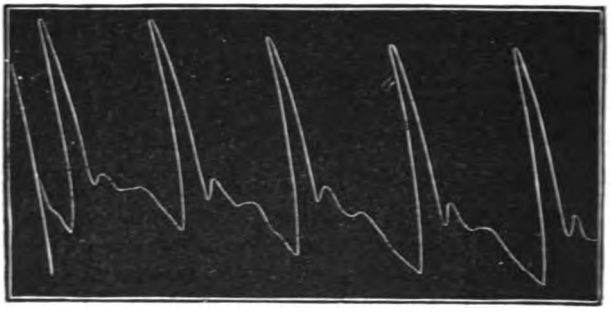

Fig. 2.—Pulse-curve in aortic insufficiency (after Strümpell).

weaker elevation due to the elasticity of the artery (*e*). The line from *e'* to *e''* indicates the secondary oscillation or elasticity of the arterial wall. Some few pathologic forms of sphygmographic tracings are characteristic. Thus, the pulse-tracings obtained in lead colic or contracted kidney show a descending elevation, with several marked elevations or smaller back-stroke elevations corresponding with increased

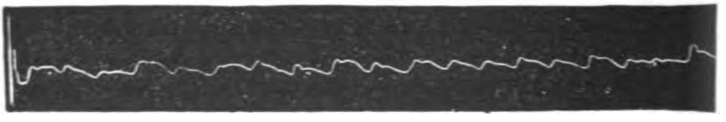

Fig. 3.—Pulse-curve with marked mitral stenosis (after Strümpell).

tension in the arterial system. The pulse of aortic insufficiency is characteristic. The apex-curve is short and pointed, and the descending line is nearly as steep as the ascending line, with marked elevations of the elasticity. There is no backward stroke elevation in this pulse, due to failure of the semilunar valves to close. The pulse-tracing of mitral stenosis shows a small, unequal, irregular, frequent pulse.

Diagnostic Value of Examination of the Pulse.—For practical purposes palpation of the radial pulse is preferable to the use of the sphygmograph for diagnosis. Sphygmography requires experience, and even then the pulse-tracings may not be accurate. In cases in which there is great doubt as to diagnosis the sphygmograph may be employed. There are many diseases in which the character of the pulse as transmitted to the finger is characteristic. In aortic regurgitation the pulse is quick, the frequency usually being slightly in-

Fig. 4.—Pulse-curve in stenosis of the aortic orifice (after Strümpell).

creased over the normal, usually regular and quicker. There are signs of great hypertrophy of the left ventricle, and the receding or water-hammer pulse is due to failure of the semilunar valves of the heart to close completely. In stenosis of the aorta the pulse is small, often of slow quality, and regular. It is not so voluminous as the pulse of aortic regurgitation just described. In mitral stenosis the pulse is small, unequal and irregular, and often rapid. In myocarditis there is often

Fig. 5—Dicrotic pulse (after Eichhorst).

a small, soft, irregular, and slow pulse. In pericarditis with effusion the pulsus paradoxus may be encountered. In febrile conditions, especially enteric fever, the dicrotic pulse is an important phenomenon.

Examination of the Veins.—In examination of the veins the jugular and cutaneous veins of the body and extremities are inspected. Occasionally, when thrombosis takes place, the deeper veins of the extremities may become accessible to examination. By means of inspection and palpation the degree

of fullness and the condition of circulation in the veins may b ascertained. Venous thrombosis also shows itself by thi method. Increased fullness of the veins may be either gen eral or local, depending upon the cause, whether it be centra or due to a peripheral part of the circulation. General in creased fullness is often due to general venous engorgement This condition is most frequently due to disease of the hear or lungs. Many of the diseases of the heart that cause an over distention of the venous system may be mentioned: Failur of force in the right ventricle; this may take place particularly in failure of compensation in which the right side of the hear is the principal chamber affected; in producing compensation especially is this true in disease of the mitral valve, mor rarely from tricuspid stenosis and insufficiency. The variou forms of myocarditis may also give rise to this condition, a may also inflammatory and other fluids in the pericardial sac in which cases the stasis may be due to pressure. Disease of the lungs that give rise to this condition are those in which the elasticity of the alveolar structure of the lungs is affected, particularly in pseudohypertrophic emphysema. If, as is very often the case, chronic bronchitis is associated with the condition, the right heart is all the more affected, and venous engorgement is apt to be the result. Exudation into the pleura may cause pressure; similar pressure may be exerted by ascites, meteorism, and large tumors pressing upward. The symptom by which this condition (engorgement of the venous system) is noted is the prominence with which the veins of the neck stand out, especially if the patient be in a recumbent posture. There may be cyanosis, edema, transudation of fluid in the serous cavities of the body, enlargement of the liver and spleen, decreased amount of urine (often containing albumin), and constipation. The local fullness of the veins is most often caused by narrowing or closure of the venous trunk, either from compression or from thrombosis. It follows, therefore, that the larger the vessel affected, the more extensive the area of abnormal fullness. Fullness of the veins of the arm points to compression of the axillary veins, most commonly from tumors or from scars following operations in the axilla. Engorged veins over one side of the chest are often encountered in mediastinal tumors. The veins under the skin of the leg are enlarged if there be thrombosis or compression of the femoral veins. Both legs may be affected as a result of compression of the inferior vena cava, or both

iliac veins, as from ascites or tumors. In engorgement of the portal vein, which occurs most often from disease of the liver, compression, or thrombosis, the cutaneous veins make up the necessary collateral circulation. Under such circumstances the veins of the abdomen are enlarged, partly extending upward toward the thorax, and partly downward toward the inguinal region. There may be a distention of veins around the umbilicus. This condition has received the name of "caput medusæ." In marked emphysema there is sometimes great enlargement of the small veins of the chest. This also results from adherent pleura and pericardium.

The Influence of the Respiratory Movements Upon Circulation in the Veins.—The influence of the respiratory movements upon the circulation in the veins shows itself in prolonged efforts at coughing, and where the return of the venous blood is artificially arrested for some time. This can be seen particularly in the veins of the neck (jugular veins). With every inspiratory movement a diminution in the size of the vein is noted, and with every expiration the vein becomes more engorged. In some diseased conditions the opposite of this is present; thus, there will be an inspiratory increase and an expiratory decrease in the size of the veins of the neck. This sign is encountered occasionally in mediastinal tumors. The pulsus paradoxus also occurs in this condition. Kussmaul has explained this as due to the traction and bending of the large vessels during inspiration.

The Venous Pulse.—Pulsation in the veins may depend either directly or indirectly upon cardiac action, and is known as the venous pulse. It may be transmitted (communicated) or actually exist in the veins. The latter condition is known as the *autochthonous* or true venous pulse. The former is usually due to the pulsation of the carotid artery, communicated to the internal jugular. The actual or true venous pulse is divided into the so-called *normal* or *negative* and the *pathologic* or *positive pulse*.

The *normal* or *negative pulse* is always *presystolic* in time, and is most often observed in the external jugular. With the systole of the heart the vein collapses. If the external jugular empties itself with the appearance of the carotid pulse and the apex-beat, and fills itself immediately, although slowly, so that it attains its complete distention before the following systole of the ventricle, it is necessarily presystolic in time. The auricle of the heart also has its share in this phenomenon.

The systole of the ventricle is the diastole of the auricle, whic favors the flow of the blood from the veins. The auricle begin to contract shortly after the beginning of the diastole of th ventricle, and thus impedes the flow of the blood from th vena cava into the auricle.

The normal venous pulse is occasionally observed in healthy persons. It is, however, small and rarely noticeable. Occa sionally, it becomes stronger, especially under circumstance of abnormal fullness of the external jugular vein.

The *pathologic* or *positive venous pulse* occurs synchronously with the carotid pulse; hence it is systolic in time. It is on of the most important physical signs in regurgitation of th tricuspid valve, being due to the right ventricle causing a quantity of blood to regurgitate into the vena cava and its adjacent branches from the right auricle, through the im properly closed tricuspid valve. It is most frequently noted in the internal jugulars. The veins of the right side of the neck show the condition more frequently and to a greater ex tent than do those of the left side. This systolic venous pulse is propagated to a certain extent into all the other veins that are given off from the vena cava, particularly to the veins of the liver, where it manifests itself by a systolic increase and a diastolic decrease in the size of the liver—"venous liver pulse." In well-marked cases of tricuspid regurgitation the systolic venous pulse is noted to a marked degree in this organ.

Diastolic collapse of the veins occasionally occurs in adhesive pericarditis. This may be associated with systolic dimpling at the apex of the heart, a bulging forward of the heart in diastole, together with the movement of the anterior wall of the chest forward, probably causing the large veins to empty themselves of their contents. This was first described by Friedreich, and is known as "Friedreich's sign."

Venous thrombosis occurs in the course of the acute infectious diseases; occasionally, it takes place in the aged. It is especially a sequel of enteric fever. It occurs most prominently in the left leg, sometimes in both legs, and rarely in the right leg alone. Adventitious sounds, usually of short duration, are at times encountered over the jugular and crural veins. They are known as murmurs. These commonly take place, as has already been indicated, in tricuspid insufficiency. The most marked sign or murmur noticed in the veins of the neck has been described as *the venous hum, venous murmur, nuns' murmur*, or *bruit de diable*. This

accompanies conditions of anemia, especially chlorosis; occasionally, though rarely, it occurs in health. It is commonly louder upon the right side. It consists in a constant regular hum. It may have a singing, piping, or musical character, and the patient may be aware of this sound.

PHYSICAL DIAGNOSIS.

The methods of physical diagnosis consist of inspection, palpation, mensuration, percussion, auscultation, and succussion. The phenomena obtained are mostly objective in nature; hence they are signs, and must be determined through clinical observation.

EXAMINATION OF THE RESPIRATORY ORGANS.

For purposes of convenience the chest may be divided into certain almost definite areas. Anteriorly, the supraclavicular fossa is noted. This is of importance, as it is the space that corresponds to the apex of the lungs. Anteriorly, the lungs reach to the sixth rib; posteriorly, to the tenth; and nearly everywhere they are in direct contact with the chest-wall, except in the neighborhood of the heart and behind a small portion of the upper part of the sternum; the apex projects from three to five centimeters above the clavicle, the anterior borders converging downward so that behind the middle of the sternum and a little to the left the lungs come closely into contact. From this point the inner border of the right lung proceeds downward to the top of the insertion of the fifth rib, gradually bending toward the right and following the sixth rib, and in the mammillary line reaching the upper border of this rib. On the left side the lung bends sharply around from the fourth rib, giving place to the heart, continuing around the fourth rib as far as the parasternal line; then running almost vertically downward, making a small bow and converging toward the right; sharply bending behind the sixth rib again, so as to cross the mammillary line under the sixth rib, being somewhat lower than the right side, and passing the axillary line between the seventh and eighth ribs and the scapular line at the tenth rib. The space above the clavicle is known as the supraclavicular fossa, the space below being known as the infraclavicular fossa. From the second rib downward we enumerate the intercostal spaces. Certain

artificial lines are drawn upon the chest: first, the median line, which is supposed to pass immediately through the middle of the sternum; second, the sternal line, lying upon the right and the left border of the sternum; third, the parasternal line, consisting of a line drawn midway between the nipple and the sternal line; fourth, the mammillary line, also called the midclavicular, a line drawn midway through the center of the clavicle through the nipple in males; fifth, the anterior axillary line, drawn through the anterior axillary fold; sixth, the midaxillary line, drawn through the middle axillary fold; and seventh, the posterior axillary line, drawn through the posterior axillary fold. These lines run parallel. Posteriorly, the spaces are known as the suprascapular space above the scapula, and the infrascapular space below the scapula. The intrascapular space lies between the two scapulæ.

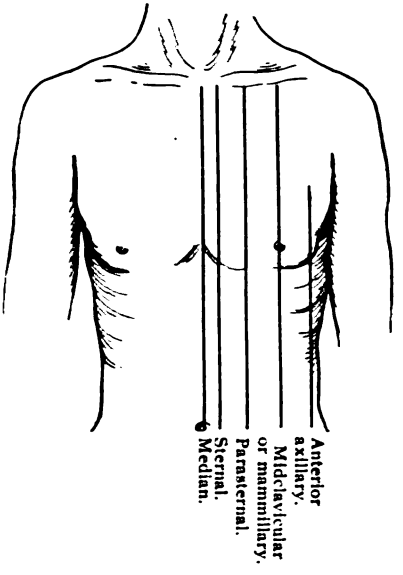

Fig. 6.—Diagram showing artificial divisions of the chest (after Eichhorst).

The methods generally employed in examining the respiratory organs consist of inspection, palpation, percussion, and auscultation; mensuration and succussion are of less importance.

Inspection.—In inspection great stress must be laid upon the shape of the chest, the respiratory movements, and the frequency of respiration. In inspecting the thorax the light should fall upon either the front or the back of the patient, depending upon the part to be examined, the examiner standing directly in front of the middle line of the body. The structure of the chest and neck in reference to peculiarities, and the respiratory movements during quiet and deep respiration, should be carefully noted. In the normal shape of the chest the most conspicuous part will be found in the symme-

try existing between both sides, although even normally there may be a slight curvature of the dorsal vertebræ toward the right side. The clavicular depressions may be no more than indicated, and the angulus Ludovici, also known as the angle of Louis (the angle formed by the junction of the manubrium and the corpus sterni), may be no more than just recognizable. The true ribs must leave the sternum with increasing obliquity from above downward, making the costal or epigastric angle almost a right angle. The normal thorax is well developed, the scapula in the upright position lying flat upon it. The intercostal spaces are only visible at the lower parts of the chest, and the dimensions of the chest and the size of the body should have a definite relation to each other. The normal number of respirations in the adults are between 16 and 20 a minute; in the new-born about 45 a minute; and at five years of age about 26 a minute. In standing or in sitting respiration is somewhat more rapid than in the recumbent posture. It is increased by psychic impressions and by exercise. For accurate counting the hand should be laid lightly upon the chest-wall. Normally, the breathing is regular, the respirations being of equal length, but under slight nervous influences they may become unequal or irregular. In inspiration the thorax enlarges, which is due to the elevation of the ribs and sternum, to the drawing of the ribs upward and downward by the intercostal muscles and by contraction of the diaphragm. In expiration the lumen of the chest is lessened. In males the type of breathing is almost abdominal, whereas in females it is almost thoracic or costal. Normally, inspiration is four times as long as expiration.

Pathologic Forms of the Thorax.—*The Emphysematous Chest or the Inspiratory Form.*—In this form there is a symmetric enlargement of the chest, and it is found frequently in chronic pulmonary emphysema. On account of its peculiar shape, it is called the "barrel chest." It is characterized by an increase of the anteroposterior diameter. It is more rounded at all points than normal, and a section of the chest would show it to be almost circular. The characteristics of this type are noticed particularly in the upper parts of the chest. The intercostal spaces are widened at the upper part of the thorax; at the lower part of the thorax the widening of the interspaces is not marked. The lower intercostal spaces occasionally are drawn in during inspiration. The ribs are at right angles to the sternum; hence the costal angle

is greater than the normal right angle and is almost obtuse. From this description it will be readily seen that the thorax is short. The breathing is typical, the expiration being markedly prolonged, and respiration generally being assisted by the auxiliary muscles of respiration. In respiration the entire chest rises and falls as if it were made of one piece. This is due to the rigidity of the chest-wall. Costal breathing does not occur.

A *unilateral expansion of the chest* arises in some forms of pulmonary disease. It may arise in the course of one-sided emphysema of the lungs, which condition occurs when the opposite lung has lost its function. In croupous pneumonia an enlargement of one side of the chest may take place, or this may occur from tumors of the lung. More often unilateral enlargement of one side of the chest arises from very large pleuritic exudations or from pneumothorax or hydropneumothorax. Circumscribed enlargement of the thorax rarely results from circumscribed pulmonary emphysema. Small encapsulated exudates from pleurisy, pneumothorax, tumors of the pleura, or empyema necessitatis give rise to this condition, as do also peripleuritic abscesses. Diseases of the heart, especially those which give rise to increase in substance, may produce circumscribed enlargement of the thorax. Similar diseases of the mediastinum may produce the same condition.

The expiratory, phthisical, alar, or *pterygoid chest* is characterized by a bilateral retraction of the thorax. It is also sometimes called the *paralytic* chest. This form of chest is almost always congenital, and occurs particularly in families that are subject to pulmonary tuberculosis. But limited respiratory effort is possible in a chest of this shape, and so it is not difficult to understand why this form predisposes to tuberculosis. Some authorities for this reason have claimed that it is not so much the lung tissue, but the shape of the chest, that predisposes to tuberculous disease. The tubercular chest is narrow and flat, especially at the upper part. The intercostal spaces are wide: the ribs incline downward from the sternum, and are bent at a sharp angle in order to come back to the vertebræ. This elongation and sloping from the sternum makes the costal angle sharp or almost acute. The shoulder-blades stand out like wings; hence the name, *alar* or *pterygoid* chest. The anteroposterior diameter is shortened.

Unilateral retraction of the thorax is due to the resorption of pleuritic exudates that have existed for a long time, and in

interstitial disease or shrinking of the lung or pleura the whole side may appear to be drawn in, so that the affected side is smaller than the healthy one. The ribs are close together, and may overlap at the lower part of the chest. The shoulder droops; the nipple and scapula are nearer the median line. The vertebral column is curved, with its convexity toward the sound side. There is diminished breathing upon the affected side or it is entirely absent, the sound side developing compensatory hypertrophy.

Funnel-shaped Chest (*Trichterbrust*).—This may occur in two ways: it may be either congenital or acquired. The acquired form is due to pressure against the lower part of the sternum along the xiphoid cartilage, and is found in many occupations, particularly among shoemakers; hence it is sometimes called "Schusterbrust"; it occurs also from mouth-breathing. The congenital form has no particular significance.

The Irregular Chest Forms.—These may be due to alteration or deformity of the skeleton. Kyphosis, a bending backward of the spine, scoliosis, a bending sidewise of the spine, or, more often still, a combination of both, kyphoscoliosis, produces an enormous deformity of the chest. These forms, however, belong particularly to the domain of surgery.

Rachitis or *rickets* produces deformities of the chest. The rachitic chest is characterized by osteoid formations near the cartilages where they join the ribs. This has been called the "ricket rosary."

The *pigeon-* or *chicken-breast*, in which the chest seems compressed sidewise and pushed forward, the ribs running sharply backward from the front so that the sternum stands out prominently like the breast of a pigeon or chicken, is a particular deformity noticed.

Harrison's grooves are depressions which merge from the *ensiform cartilage* toward the axilla; the curve is transverse, and may deepen with inspiration. It is often associated with the rachitic chest.

Respiratory Type.—This has already been indicated in saying that males have the abdominal and females the costal type. Occasionally, there is a mixture of these two types, known as the costo-abdominal type. Movement of the diaphragm may be noticed in thin persons. It shows itself as a light shadow, taking place particularly upon the right side, with each inspiration falling downward, and with each expiration moving upward. The breadth of the excursis is from

five to seven centimeters. Litten has called this the *diaphragm phenomenon*, and believes it is of great importance to determine the lower border of the lungs. Changes in the respiratory types may occur: that is to say, the costal type may be observed in men, especially where there is disease of the abdominal organs or the diaphragm. The abdominal type may be seen in women in whom there is disease of the thoracic organs.

Inspiratory Retraction of the Intercostal Spaces.—If the normal respiratory movements are carefully noted, it will be seen that in quiet breathing, during inspiration the intercostal spaces flatten and come forward only to the anterior edge of the ribs. No movement beyond this is seen in health. From the fourth rib downward, at the beginning of inspiration a decided deepening in the intercostal spaces may be observed. Pathologic inspiratory retractions occur in diseases in which the atmospheric air does not gain entrance into the alveolar structure of the lungs. This may be due to mechanical causes in which obstruction may prevent entrance of air, such as mucus, pus, blood, fibrinous exudates, tumors, foreign bodies, inflammation of the mucous membranes, spasm of the bronchial muscles, or disease of the alveolar structure itself. This is noticed most prominently in children, and is seen in diseases like laryngitis, diphtheria, and croup. If both sides of the thorax show retraction, the difficulty is most apt to be in the upper air-passages, in the larynx, or in the trachea. Retraction of one side of the thorax is found when the principal bronchus of the side is affected. This may be due to foreign bodies, inflammation, collection of mucus or inflammatory exudates, compression or narrowing, from lymphatic glands, mediastinal tumors, aneurysms, or large pericardial or pleural exudations. Circumscribed retractions are due to local disease, such as the plugging of several small bronchi or alveoli of the lung. This occurs frequently in tubercular disease, and is usually bilateral. Expiratory bulging of the interspaces may take place from narrowing or closure of the glottis, such as may result from vomiting, coughing, or asthma. Circumscribed bulging is noted in pulmonary emphysema, giving the chest the appearance already described as the inspiratory form.

Intensity of the Respiratory Movements.—Sibson has called attention to the fact that the right side of the chest expands slightly more than the left. This condition has been attributed to the greater development of the muscles upon the

right side, the greater width of the right bronchus, and the larger size of the right lung. In sleep the respiratory movements are slightly less frequent. Bodily exercise and psychic influences produce an increase in the respiratory functions. These conditions are all considered as physiologic. Diminished respiratory movements are seen in pulmonary emphysema and in tubercular disease of the lungs, but if complication occurs, such as bronchitis, an increase in the respiratory movement may be seen. In fainting (syncope) diminished respiratory movements are noted. A unilateral diminution in the respiratory intensity may be observed in diseases of the bronchi, if the free access of air in reaching the alveolar structure of the lungs is prevented. This is especially noticeable if foreign bodies gain entrance to the air-passages. The same condition is often associated with disease of the alveolar structure of the lung, especially if the alveoli contain inflammatory masses or the lung is the seat of miliary tuberculosis. In disease of the pleura this condition is observed. Increased frequency of respiratory intensity takes place in all cases in which the change between oxygen and carbonic acid is hindered. Disturbances of respiration, due to diseases of the heart, associated with stasis in the pulmonary circuit, and disease of both of the large bronchi may cause the condition. In paralysis of the phrenic nerve, in inflammation of the pleural or peritoneal lining of the diaphragm, from meteorism from tumors or from accumulation of fluid in the abdomen, the condition may occur.

The Respiratory Rhythm.—In health there is a regular change in the contour of the chest between inspiration and expiration. This is known as the respiratory rhythm. Diminished frequency of respiration results from many diseases of the brain and its membranes; hence the condition is found in meningitis from pressure of tumors or large hemorrhages (being associated with some dullness of intellect), even passing into the so-called "Cheyne-Stokes respiration." Diminished breathing further arises in the acute infectious diseases, and finally it is always noted in the death agony.

Cheyne-Stokes Respiration.—In this form, a group of respirations alternate in intensity and rapidity regularly, with a more or less well-defined and prolonged pause. A pause in respiration is known as *apnea*. The change from one condition to the other is gradual. Beginning and terminating with shallow breathing (the patient in the majority of cases being in coma), the respirations have a snoring or snorting character.

The pause in the respirations may last from a few seconds to a minute or longer. The number of respirations that occur before the pause also vary. Most frequently there are from eight to twelve. Besides the well-marked form just described, there are some less striking varieties. Occasionally, the pause or apnea may be missing, and the respiration may not be so deep. This form of respiration is very likely to be observed in diseases of the brain, from disturbances of the circulation, or from toxic conditions. Cheyne-Stokes respiration is always a symptom of exceeding gravity. The explanation of this form of peculiar breathing has not been satisfactorily determined; it is sometimes accompanied by cyanosis. The duration of the Cheyne-Stokes respiration varies considerably. It may last for days or even for weeks. Cases have been recorded in which the duration has been noted for seven months.

Difficult Breathing, or Dyspnea.—If the respirations be labored, whether the number be normal, prolonged, or more frequent, the condition is spoken of as *dyspnea*, especially if the breathing is labored or rapid so that there is an interference with the exchange of gases in the pulmonary circuit. It may also be due to an increased formation of CO_2, occurring in fevers. Dyspnea occurs in stenosis of the upper air-passages, from intratracheal tumors, foreign bodies, inflammation, cicatrices from ulcers or strictures, and from paralysis of the laryngeal muscles, which produce a narrowing of the air-passages. Dyspnea often occurs from diseases of the brain, and the acme or height of the Cheyne-Stokes respiration is practically dyspnea. Expiratory dyspnea takes place commonly in bronchial asthma, in pulmonary emphysema, and in disease of the diaphragm. Ordinarily, the dyspnea is both inspiratory and expiratory. Dyspnea is a common symptom of fever, especially in persons of neurotic temperament. It occurs in diseases that are accompanied by pain, such as croupous pneumonia, pleurisy, inflammations of the diaphragm or peritoneum, fracture of the ribs, muscular affections of the thorax, disease of the bronchial tubes, particularly those that show a tendency to narrowing or stenosis, as has already been pointed out in asthma, occasionally in forms of bronchitis (croupous bronchitis), and in bronchopneumonia. Various diseases of the lungs give rise to the condition, such as pneumonia, edema, infarcts, emphysema, tuberculosis, pleurisy with effusion, pneumothorax, tumors of the lung and pleura, abdominal affections that show a tendency to push the diaphragm upward, in de-

formities of the chest, such as kyphoscoliosis, paralysis of the respiratory muscles, as in tetanus and epilepsy, and is sometimes due to disease of the heart in which the pulmonary circuit is interfered with, particularly in disease of the left heart. When dyspnea becomes extreme, it is known as *orthopnea*. The symptoms of this condition are usually great anxiety, a peculiar expression of the eyes, protrusion of the eyeballs, cyanosis, cold sweat, and exceedingly rapid respiration, so that the patient must often assume the erect posture.

Palpation of the Respiratory Organs.—This can only be carried on indirectly, as obviously only the thorax and the neck, which corresponds to the beginning of the trachea, may be directly palpated. On the one hand palpation confirms the results of inspection; it has also an independent value, as some signs are discovered upon palpation that can not be noted upon inspection. In palpation we notice first the movements of the chest, the resistance, the presence of pain upon pressure, either localized or general, fluctuations of the chest, the vocal fremitus, friction fremitus, and palpable rhonchi. Palpation of the respiratory excursis should be tested by laying the hands flatly upon each side of the chest, first anteriorly and then posteriorly. In this way it will be noticed whether one side of the chest expands better than the opposite. It is also well to place the hands posteriorly in the axillary line at the bases of the lungs, in order to notice the expansion in this region. The greatest changes in resistance may be found in the young, in whom the thorax is compressible. Quite the opposite is true in the aged, as great resistance is here encountered upon attempts at compression. This is due to the calcareous change of the cartilages of the ribs. Pathologically, there is an early tendency to greater resistance of the thorax in tubercular disease of the lungs. This sometimes is not without its value in diagnosis. The emphysematous chest shows great resistance upon attempts at compression, as does also the rachitic thorax. Pain upon palpation is of great value in diagnosis in diseases of the chest. The painful region may be mapped out with some degree of accuracy by feeling each intercostal space with the finger, and determining which one of them is tender upon pressure. In this way it may be determined whether the pain radiates or not. Pain is found in all diseases of the chest in which the pleura is implicated; thus, it is common in the first stage of pleuritis, in croupous pneumonia, and in pulmonary tuberculosis in

which the pleura is involved. It must also be noted whether pain is due to involvement of the rib, from caries, or from fracture. Myalgia of the chest muscles may also produce pain, especially in the superficial muscle. Pleuritic pain and intercostal neuralgia are sometimes quite difficult to differentiate. (The differential diagnosis between these two conditions has been given under Pleurisy.) Abscesses within the chest or in the pleura may also cause pain from pressure. Fluctuation upon palpation in the region of the thorax is rare. It may occur from diseases of the lung in which pus has bored its way through to the chest-wall, or from the breaking through of an empyema (empyema necessitatis). An important sign to determine whether the fluctuation be due to disease of the lung or pleura or to external lesions will be found in the fact that slight pressure may cause the fluctuation to disappear, and coughing reproduce the fluctuation, these signs indicating intrathoracic disease.

Vocal or Pectoral Fremitus.—If the flat hand be laid upon the thorax and the voice be used, a peculiar quick trembling or vibration will be communicated to the hand with the beginning of the first spoken word and will cease with the use of the voice. This palpatory phenomenon is known as *vocal*, *pectoral*, or *tactile fremitus*. It is produced by the vocal cords being thrown into vibration, the sounds produced being carried down from the larynx into the thorax, through the bronchi into the alveolar structure of the lung, and thence communicated through the chest-wall to the palpating hand. The following conditions in reference to vocal fremitus must be noted: The louder the voice is used, the more powerful and distinct the vocal fremitus. The vocal fremitus is proportionately plainer the deeper the voice. As males usually have a louder voice than females, the vocal fremitus is commonly stronger in the male than in the female sex. It follows that when the whispered voice is used, the vocal fremitus is very much diminished or even entirely absent. It must be further noted that vocal fremitus upon the right side at the apex of the lung is more distinct than upon the left. This depends upon the fact that the right bronchus is larger and is more superficially situated than the left. Finally, vocal fremitus depends upon the structure of the thorax—in a thin thorax with very little adipose tissue vocal fremitus will be more distinct and stronger than where the chest is developed by muscle and cutaneous fat. The lower the pitch,—that is

to say, the slower the vibrations,—the more distinct the vocal fremitus. In children the vocal fremitus is not so distinct, as the vibrations of the voice are too rapid.

Pathologic Changes in the Vocal Fremitus.—Vocal fremitus may be increased, diminished, or absent. Diseases of the pleura usually decrease the vocal fremitus. Diseases of the lung, on the other hand, usually give increased vocal fremitus; however, many circumstances may influence these conditions. In consolidation of the lung the vibrations are transmitted with greater force to the hand, producing increased vocal fremitus; thus, we have an increase in pneumonia, tuberculosis, and hemorrhagic infarct. Two conditions may influence this: if the main bronchus of the consolidated part be occluded, the vocal fremitus will be absent, as vibrations can not reach the part; or if a pleural effusion be present over the same area, vocal fremitus is apt to be diminished or absent, as fluid does not conduct sound so readily. Diminished vocal fremitus results from any cause that increases the distance between the lungs and the surface of the chest, interfering with the conduction of vibrations; thus we have diminished vocal fremitus from thickened pleura and from small pleural effusions. If the collection of air in the bronchi is lessened from a diminution in the caliber of the tube, as may occasionally occur in emphysema or asthma, or if there be pulmonary cavities filled with fluid, vocal fremitus is diminished. Absent vocal fremitus is produced by occlusion of the bronchus from external pressure, such as tumor, aneurysm, or enlarged gland, or from an accumulation of large quantities of air or fluid in the pleura; hence vocal fremitus is absent in large pleural effusions, pneumothorax, hydropneumothorax, and allied conditions. The same condition practically exists when the pleura is greatly thickened. Marked dilatation of the bronchial tube, especially if superficially situated, produces increased vocal fremitus. This may occur from bronchiectasis. The same is true of a pulmonary cavity, superficially situated and empty. There is a sound occasionally produced by the passage of air through fluid in the bronchial tubes that is transferred to the hand when laid upon the chest. These vibrations are known as *rhonchi*. They occur particularly during inspiration, and take place in such diseases as bronchitis and asthma. They may be either coarse or fine. Occasionally, there are localized areas of vibration in pulmonary tuberculosis. This is due to

the passage of air through fluid in the cavity. In bronchitis of children, rhonchi are very apt to occur. The tracheal rale occurring in the death agony is an example of a rhonchus. Vibrations may be transmitted to the hand from inflammation of the pleura. They are often felt only during inspiration, but may occur during inspiration and expiration, and rarely only during expiration alone. At the bases of the chest, either anteriorly, posteriorly, or laterally, they are increased by deep inspiration, and are not influenced by coughing. Rhonchi, on the other hand, may disappear entirely upon coughing or deep breathing. Occasionally, upon palpation, if the hand is placed over a large cavity containing air and fluid, the splashing (the equivalent of succussion) is felt. This takes place almost exclusively from hydropneumothorax. Under rare circumstances, if there is a large pulmonary cavity partially filled with a thin secretion, a similar sound may be noted. Crepitation is sometimes transmitted to the hand upon palpation. It results from subcutaneous emphysema. The same condition is noted from prolapse of the lung, but this is a surgical disease. Finally, *pulsation* of the thorax takes place upon palpation. If the pulmonary tissue in the immediate neighborhood of the heart becomes airless, the cardiac systole is transmitted to the hand upon the left side at the base in the form of pulsation. Graves first called attention to this in hepatization of the lung in croupous pneumonia. Carcinoma may produce the same condition. In left-sided empyema, and especially from empyema necessitatis, pulsation is transmitted to the hand, known as "pulsating empyema." Rarely, pulsation may occur upon the left side from a serous exudate, Traube and Frantzel having called attention to this fact. Peripleuritic abscesses in the neighborhood of the heart may also give rise to pulsation upon palpation.

Mensuration of the Thorax.—The measurement of the diameters of the chest has but slight diagnostic value, principally because the individual variations are so great and the eye is usually sufficiently competent to estimate the differences better perhaps than any instrument. Cyrtometry has for its purpose the determination of the shape of the chest. For this purpose pliable media, usually of lead, are used, which are molded around the chest, and afterward removed and drawn upon paper. This method gives but limited results; hence mensuration is not in general practical use.

Percussion of the Respiratory Organs.—Percussion has for its object the determining of the physical condition of the respiratory organs. This is done by means of striking upon the chest. The method was first used about the year 1761 by Auenbrugger, a Viennese physician, and was further improved by Corvisart, the physician of Napoleon, being still further studied by Skoda and Traube. Two conditions are noted upon percussion: (1) that *sounds* are produced and (2) *resistance* is determined. The sounds produced may either produce a musical tone, or they may be nothing more than a noise, the vibration of which can not be definitely determined, there being no uniformity. Sounds have certain well-defined characters: they consist of *pitch*, *volume*, *duration*, and *quality*. The pitch is due to the rapidity of the vibrations. It may be either high or low, as the vibrations are rapid or slow. The volume (intensity) depends upon the amplitude of the vibration, and varies directly as the square of the amplitude of vibrations. It is further modified by the amount of force used in producing the sound. *Duration* simply means the period of time taken up in the production of sound. Notes or sound, high in pitch, are of diminished volume and of short duration. Sounds low in pitch are of increased volume and of long duration. The three conditions just described determine the *quality*. *Clearness* is the term used to designate sounds without the character of tones or notes. They are of *good volume*, *long duration*, and *low in pitch*. A *dull* sound is of high pitch, of small volume, and of short duration. Irregular sounds or noises in which pitch, volume, or duration can not be definitely determined are described as *flat;* absolutely flat sounds are characterized by entire absence of air. The dull and clear sounds are alone produced over structures containing air. Areas in which there is a definite relation between air and solid material produce sounds that vary between clearness and dullness. *Resonance* and *tympany* are modifications of clear sounds.

Method of Percussion.—The thorax may be tapped directly by the hand or finger. This method is known as *immediate* percussion, and was the method originally employed. It is, however, no longer in general use. The general practice consists of employing some medium to intervene between the surface of the chest and the instrument used for striking or percussing. The medium has received the name of *pleximeter*, and the method is called *mediate percussion*. The pleximeter

may consist of a small piece of bone or ivory, sufficiently small to fit between the ribs, or, what is better, the fingers of the hand may be used for this purpose. The instrument used in striking to produce the sound is called the *plexor*. This may be a small hammer of moderate weight, with a slightly flexible handle, consisting of a metal or rubber mallet. Far better for this purpose are the fingers of the hand, as with them an idea of resistance is also obtained, which is impossible in the use of the pleximeter and plexor.

Method Employed in Percussion.—The pleximeter or finger must be placed in direct apposition to the surface of the chest, and no air must intervene between the medium and the chest-wall. The finger should be placed *parallel* with the ribs, *but should not cross them.* Corresponding areas of the chest upon the right and left side should be percussed alternately. The use of the finger as plexor requires some practice. Ordinarily one finger, preferably the middle one, is used, but sometimes two fingers are employed. The finger should be bent at right angles and retained in a fixed position, and it should strike the pleximeter perpendicularly to its plane (at right angles). The force of the blow should be regular and even, and must come *from the wrist alone.* Anteriorly, percussion should be begun in the supraclavicular fossa, proceeding downward from interspace to interspace, and, as has already been stated, comparisons being made with the opposite side. Next, the axillary portions and, finally, the posterior portions of the chest should be examined in the same way. To perform percussion properly, the patient should be in the erect posture, with the arms held loosely at the sides. No position should be chosen that would draw the chest muscles into contraction. When the posterior aspect of the chest is examined, it is well to have the patient fold his arms and bend slightly forward. This separates the scapulæ more widely and allows more freedom of access to the interscapular spaces. It is well to remove all clothing if practicable. A thin, loose garment may be used from motives of delicacy, or only parts of the chest should be exposed if there is any possibility of chilling the surface of the body.

Normal Percussion.—Three qualities of sound are developed over the normal thorax by percussion. Tympany is elicited over the trachea, resonance over the lungs, and dullness over the cardiac area. By *resonance* is meant a clear sound produced over the normal lung tissue, being due to the

vibration of the chest-wall and of the air in the various bronchial tubes. The term normal pulmonary resonance or vesicular resonance is used to designate the same condition. The note called resonance can scarcely be described as a tone, as the relation of air being confined in innumerable small sacs in the solid structure of the lungs prevents tone formation, and the vibrations do not occur in unison. It is preferable to use the term clearness or resonance. Normal pulmonary resonance is developed at various parts of the chest. Its most defined type is encountered in the upper axillary region behind the angle of the scapula, and on the anterior surface of the chest in the second interspace. It is somewhat higher in pitch at the right apex than at the left. Accumulation of adipose tissue and thick muscular chest-walls impair resonance, and require deeper percussion. The elasticity of the chest also bears some relation to the quality of the note encountered. In old age with rigid chest-walls the note is less clear than in children. It should be remembered that in percussing upon the left side of the chest below the third rib dullness is elicited, which is due to the presence of the heart. If the note be more than clear, having a higher pitch, it is usually due to an excess of air, the condition being called *hyperresonance*. If the note be less clear, there is a diminution in the amount of air, or an increase of the solid structure, and it is spoken of as *impaired resonance*. *Tympany* is present in percussion over a cavity with smooth walls containing air, in which the sound is low in pitch, of considerable volume, and of long duration. In health it can only be elicited over the trachea, over the stomach when it is empty, and over the large and small intestines. It sometimes possesses a metallic quality. Tympany may sometimes, though rarely, be elicited normally over the posterior portion of the lungs in children and infants. It differs from resonance in being lower in pitch, smaller in volume, and shorter in duration. For practical purposes it may be stated that resonance comes midway between tympany and dullness. The larger the amount of air present, the greater the tympany. The sound in percussing over the heart is termed dull. It shows the absence of air. It is necessary in percussion to estimate the pitch of the sound elicited. This is quite difficult to do, and requires considerable practice. Under normal circumstances, the sound under the right clavicle is higher in pitch than the sound under the left, but it can not be spoken of as being dull in character.

Generally speaking, a high-pitched note is considered a dull note. Change in pitch makes it possible at times to outline particular organs.

Degree of Resistance.—This can only be ascertained by the sense of resistance communicated to the finger. Over organs containing air, the resistance is slight. The sensation imparted to the finger is as if the parts recede and yield. As the quantity of air becomes less and less, and approach is made to solid structure, resistance becomes proportionately increased, until with an entirely airless structure it is great. Finally, a difference must be made between superficial and deep percussion. In superficial percussion a note is obtained of that portion lying directly beneath the part percussed. Superficial percussion is light percussion. In deep percussion the blows are delivered with force, and the sound of structures deeply situated is elicited in this way. By *auscultatory percussion* is meant that percussion is carried on while the stethoscope is placed at some slight distance. In this way the borders of an organ may be outlined. Mediate percussion may be used for this purpose. Percussion has for its object the determination of the size of the lungs, the presence or absence of abnormal sounds so that the physical condition may be ascertained, and the apparent size of organs in the chest noted (topographic percussion). The normal boundaries of the lungs have already been given. An increase in size takes place in pulmonary emphysema. A diminution in size may occur at one or both apices, as in pulmonary tuberculosis and in adherent pleura, and also in other diseases of the lung.

Abnormal Percussion Sounds.—Disease of one or both apices is recognized by impaired resonance. If the resonance at the left apex should be as high in pitch as that at the right, it would show implication of the left apex. Dullness is due to the formation of solid new growths in the air structure of the lung, or to the development of fluids in the air-cells and tubes. It may also be due to the medium that obscures the sound of the lung between it and the finger. It occurs in diseases like croupous pneumonia, bronchopneumonia, tuberculosis of the apices, large hemorrhagic infarcts, and solid tumors. Under the second heading the pleural exudate plays the chief rôle. This is found at the lower part of the chest, and dullness or, more correctly speaking, flatness, occurs as high as the level of the fluid. If the amount of the effusion be very large, it may even extend to the apex. A transudate into the pleural cavity also produces

flatness at the base (hydrothorax). Great thickening of the pleura also produces impaired resonance, and it is sometimes very difficult to determine between a small pleural exudate and a thickened pleura. Finally, in processes that thicken the chest-wall, such as tumors or edema, impaired resonance or even complete dullness or flatness is produced. In consolidated lung and in fluid in the pleural cavity above the level of the fluid and above the level of the consolidation a high-pitched, somewhat tympanitic (hyperresonant) note is encountered called *Skodaic resonance*. The note is short, the tone is clear, and the pitch is raised. Skodaic resonance is due to consolidation or an accumulation of air above the layer of fluid. The tympanitic sound occurs abnormally if there is an increased amount of air in any part of the lung or bronchial tube. It may result from retraction of the lung, or from shrinkage due to any cause; hence it may take place from pleuritic exudation, tumors, pericardial effusion, rarely from hypertrophy and dilatation of the heart, or from pressure beneath the diaphragm. It is known as relaxation of the lung tissue. Marked shrinking or thickening of the lung produces a tympanitic note like percussing over the trachea. This has been called "Williams' tracheal sound." It takes place from pulmonary cavities (vomicæ). If the cavity is empty and is superficially situated and communicates with the bronchus, the "cracked-pot" sound is elicited. To produce this the patient should have the mouth open, and the area in which the cavity occurs should be sharply and firmly percussed. The sound may be imitated by placing the two hands lightly together and striking them against the knees. If the cavity is large, with smooth walls, a metallic quality may be added to the tympanitic resonance. If the cavity is covered by thickened lung tissue or pleura, a note of decided tympanitic quality is encountered. This is sometimes spoken of as dull tympany. As the cavity becomes filled with fluid the tympanitic note gradually gives way to dullness or flatness. The tympanitic sound becomes more distinct, louder, and higher in pitch if the patient opens the mouth wide. This is noted particularly in cavities that communicate with the bronchus. This is known as "Wintrich's change of sound." Finally, the tympanitic note occurs in pneumothorax. An abnormally loud and deep sound takes place in severe pseudohypertrophic emphysema. In pneumothorax the tympanitic note often has a metallic quality added. Flatness occurs only over organs

entirely devoid of air, such as the liver, or in percussion over the thigh and over extensive accumulations of fluid.

Auscultation of the Respiratory Organs.—This is one of the earliest methods of examination, Hippocrates having described certain auscultatory phenomena, particularly the succussion sound. Pleuritic friction and certain rales appear to have been known in ancient times. The method, however, was neglected until about the beginning of the nineteenth century, when Laennec, about 1819, called attention to auscultation by his thesis published in Paris. The development of auscultation, as in fact nearly every method of physical diagnosis, we owe particularly to the studies of the Viennese physician, Skoda. As in percussion, so in auscultation, there is an immediate and a mediate method. In the first method the ear is laid directly against the chest-wall, whereas in the last method an instrument known as the stethoscope is used. Laennec himself practised the stethoscopic method. In auscultation without the use of the stethoscope the phenomena appear louder, and a larger part or portion of the thorax can be examined than by means of the stethoscope. This should be remembered in the examination of feeble and weak individuals in whom a rapid examination of the chest is necessary. The stethoscope becomes of special value when it is necessary to localize certain auscultatory phenomena. It is of especial importance in the examination of the heart and the large vessels. It is furthermore noteworthy that there are several portions of the chest that are not accessible to the ear directly, and that can only be examined by the stethoscope. The supraclavicular fossa, the examination of which is especially important in tubercular diseases of the lungs, is an instance of this. There are decided disadvantages in the use of the immediate method, as in the examination of unclean, perspiring patients, those affected with skin diseases, etc. In such cases it is better to use a towel. Care must be taken that there are no folds in the towel, and the ear must be placed against the thorax. Auscultation is valueless if it is undertaken over a number of garments, as so many extraneous sounds are produced as to render the value of the method unimportant. From what has been said it may be gathered that it is impossible to do without stethoscopic auscultation. Stethoscopes have been improved ever since the method of auscultation came into general use. At first they consisted of a solid piece of wood. These were used by

Niemeyer and Quincke. But to-day they are either the double stethoscope, after the model of Camman, or the single stethoscope. In using it it is well to remember that the instrument should be placed on the bare chest and not over clothing. In auscultating the chest the following points must be carefully noted: the character of the respiratory murmur, which may be of a vesicular, bronchial, or mixed type; the presence of rales, whether dry or moist; the presence of pleural friction sound, and the auscultation of the voice.

Vesicular Breathing.—In auscultation of the sound lung, at almost all parts of the thorax, normal vesicular breathing is noted. It is heard almost exclusively in inspiration, whereas in expiration an indistinct or slight blowing sound, which is more of the character of bronchial breathing, is noted. The expiration is lower in pitch than the inspiration, but not so loud. Vesicular respiration may be imitated by almost completely closing the mouth and leaving only a small cleft, and then with some force drawing air into the buccal cavity; or the lips may be put in position as if to pronounce the consonants "v" and "f," and air drawn into the mouth. This illustrates very well the character of normal vesicular breathing. The character of its production is by no means settled. Laennec suggested that it occurred from friction of the inspiratory stream of air against the wall of the bronchial tubes and the infundibula of the lung. Several other theories have been given for the production of this sound, the most likely of which, however, are those advanced by Baas and Penzoldt, that vesicular breathing is produced by the column of air entering through the larynx and into the bronchus, producing bronchial breathing, and which, traveling further through the lungs, loses its bronchial character as the sound becomes disseminated and is changed into the vesicular murmur. The recognition of vesicular breathing is of importance. It shows that the alveoli and the finer bronchi are still capable of admitting air. Care must be taken, however, to remember that in certain diseased conditions of the lung a few air vesicles may yet be capable of receiving inspired air, and thus the vesicular murmur be produced. This sometimes takes place in bronchopneumonia and in miliary tuberculosis. In superficial cavities in which, as a rule, bronchial breathing is heard, vesicular breathing may occur, which can not be explained in any other way than that the alveolar structure surrounding the cavity is still capable of admitting air. The following points must be

noted in reference to vesicular breathing: the *force*, the *strength or intensity*, and the *interrupted vesicular breathing*. The strength of vesicular breathing depends upon age and sex. Children and women usually show more pronounced vesicular breathing than men. This depends particularly upon the smaller lumen of the larynx, giving a higher pitch than the larger male larynx. In old age vesicular breathing is often found to be intense. In general miliary tuberculosis and in edema of the lungs strong, high-pitched vesicular breathing is often noted.

Strength and Intensity of the Vesicular Murmur.—In superficial respiration the character of the vesicular murmur is often indistinct and not at all well characterized. The intensity of the vesicular murmur in health then depends upon the force of the inspiratory movement and the thickness of the thorax. Deep inspiration and coughing or crying, especially in children, show a well-developed type of the vesicular murmur. In Cheyne-Stokes respiration the more superficial the respiratory movement, the more indistinct is the vesicular murmur; whereas the deeper the respiration, the more developed the vesicular murmur becomes. The thickness of the walls of the thorax has an important influence upon the character of the vesicular murmur. Thus, in well-developed muscular chests the vesicular murmur is not pronounced except upon very deep inspiration. In thin chests, on the contrary, the vesicular murmur is often well marked. In the erect posture it is often more pronounced than in the recumbent posture. After meals and after moderate exercise the vesicular murmur is more distinct, whereas in sleep and in weak individuals it is not well characterized. Laennec called attention to the fact that from the use of tight corsets the vesicular murmur becomes stronger at the apices; women therefore show a stronger and more pronounced vesicular murmur than men. As a rule, the vesicular murmur is better heard upon the left side of the chest than upon the right. Normally, the vesicular murmur is stronger upon the upper anterior surface of the chest. It is less distinct posteriorly and in the axillary regions. It is loudest directly beneath the clavicles. It progressively diminishes in intensity as the lower parts of the thorax are reached. It is furthermore noteworthy that in the spaces between the parasternal and mammillary line the murmur is more intense than at the sternal border and in the neighborhood of the axillary line. Over the sternum

itself the vesicular murmur is plainly heard, due to transmission. It is better marked at the upper parts than at the lower parts of the sternum. In the axillary line the murmur is more intense at the upper than at the lower parts of the chest. In the scapular regions the murmur is not well developed; this is particularly due to the great thickness of the muscles. When the vesicular murmur becomes stronger and more sharply defined, it may be imitated by placing the lips in the position of pronouncing the consonant "f," and drawing air into the buccal cavity, which is the usual form of respiratory murmur heard in children; this is known as *puerile respiration.* It is of higher pitch than the normal vesicular murmur found in adults, and it is found occasionally in chests with very thin muscular walls. Diminished vesicular breathing occurs from narrowing of the lumen of the bronchial tubes themselves, inflammation of the mucous membrane, fibrinous exudates, tumors, foreign bodies, or compression of the tubes. The vesicular murmur is further diminished, or may be entirely absent upon the surface of the thorax, by accumulation of fluid or gas in the pleural cavities. Diseased conditions of the walls of the thorax, such as edema, or well-developed adipose tissue may give rise to a diminution in the respiratory murmur. Diseases that produce pain and in which breathing is diminished may show a lessening in the respiratory murmur, such as pleuritis, myalgias of the intercostal muscle, thickening of the pleura; and it is particularly found in chronic pulmonary emphysema. In the latter case the diminution of the respiratory murmur is due to the loss of elasticity in the alveolar structures of the lung. Diseases of the upper air-passages may also produce diminished or absent vesicular murmur: diphtheria of the larynx, paralysis of the posterior crico-arytenoid muscles, and paralysis of the muscles of one side of the chest are examples. Pathologic increase of the vesicular murmur is invariably associated with an increased and quickened respiratory movement. It is often found in nervous and hysteric women. It is almost invariably associated with febrile diseases. In all conditions in which one lung is entirely or for the most part diseased the opposite lung takes on extra function. In such a case an increased vesicular murmur is noted, which is spoken of as *vicarious*, *supplementary*, or *compensatory breathing.* Bronchitis often increases the intensity of the vesicular murmur. A sign of great diagnostic importance is the increased and more powerful vesicular murmur of

the apices of the lung, this condition being very commonly associated with pulmonary tuberculosis. This sign is of especial importance should it occur at both apices. Wintrich first called attention to a systolic vesicular murmur. This is frequently found in normal healthy persons at the margin of the lungs where they come in contact with the heart. It follows from this that it is more frequently observed upon the left than upon the right side. It is an acoustic phenomenon that the vesicular inspiratory murmur is rhythmically strengthened with the systole of the heart, and is decreased or even becomes inaudible with diastole. Interrupted vesicular murmur shows itself through a break in the continuity of the inspiratory sound. It is occasionally heard in children from fear, especially during an examination by the physician. It is encountered during the period of chill, and often takes place in painful conditions of the pleura and thorax wall; this has been termed *jerking respiration.* Its diagnostic importance consists in the fact that it occurs in catarrhal conditions of the finer bronchi, especially at the apices of the lung, and is an early sign of the infiltration of tubercles ("cog-wheel respiration"). This sign is especially important if it should take place at both apices. *Jerking inspiration* occasionally occurs in severe bronchitis. It is often accompanied by prolonged expiration.

Vesicular Breathing with Prolonged Expiration.—In healthy persons inspiration is, as a rule, four times as long as the indistinct expiratory murmur. However, should inflammatory exudates appear in the bronchial tubes, the expiratory murmur sometimes equals, and occasionally is longer than, the inspiratory murmur. Should this sign be localized to the apices, it is indicative of incipient tubercular disease. Prolonged expiration further occurs in bronchial asthma and in chronic pulmonary emphysema, as in both these diseases expiration is of itself prolonged.

Bronchial Breathing.—This type of respiration is heard normally in listening over the trachea and occasionally in the intrascapular spaces; for this reason it is sometimes called *laryngeal* or *tracheal* breathing. It is well for the student to familiarize himself with this sound by placing the stethoscope over the trachea. It will be noted that the expiratory murmur is decidedly stronger than the inspiratory. It is of higher pitch and has a blowing quality, being especially marked in expiration. When this particular variety of breathing is heard

over any other portion of the lung, it indicates some pathologic process. Bronchial breathing is encountered over airless spaces in the lung tissue and over cavities. It occurs over airless lung tissue because only tissues containing air have the property to change the bronchial sound as it passes through the trachea and bronchi into the vesicular murmur. If, then, the parts of the vesicular structure of the lung become impervious to air through consolidation from any cause, the property of changing the bronchial murmur of the larynx to the vesicular murmur disappears, and the bronchial respiratory sound is heard in the affected area. The production of bronchial breathing in cavities is due to other causes. Should a bronchus terminate in a cavity with firm walls, the transmitted sound from the trachea enters directly, producing columns of vibrations that are heard in the cavity as bronchial breathing. This may occur both in inspiration and in expiration. In this case the bronchial respiratory sounds are produced in the cavities themselves. In the bronchial breathing over the cavity it may be noted that the sound is more intense than the bronchial murmur produced over the trachea, and it occasionally differs in that it may be louder in inspiration than in expiration. This has also been called *cavernous breathing*. Two things are necessary for the appreciation of bronchial breathing—(1) that the affected area must be superficially situated, and (2) that the bronchial tubes that lead to the diseased part must be free to the egress of air; thus it may occur in consolidated portions of lung, as in croupous pneumonia. The bronchus that leads to the pneumonic area may be plugged by mucus or other secretion, and bronchial or other forms of respiration will be entirely absent. The same condition takes place if a pleural exudate should be present in the affected area. Bronchial breathing is more easily appreciated by the ear than is the normal vesicular murmur, as the sound more closely resembles a musical note, being decidedly higher in pitch than the normal vesicular murmur. The pitch of bronchial breathing occurring in the trachea is higher in inspiration than in expiration, being also changed by opening and closing the mouth. It is higher in children and in women than it is in men. Over cavities the pitch of the bronchial murmur will depend upon the fact that the smaller the diameter of the cavity and the larger the opening into the bronchus, the higher the pitch of the respiratory sound will be. Occasionally, there may be a mixture of the

bronchial and vesicular types. This is known as *bronchovesicular respiration*, and shows the quality of both the bronchial and vesicular types. One or the other of the types may predominate. It is also known as *harsh respiration*. The more pronounced the bronchial element, the higher the pitch and the more prolonged the expiration. The important element in the recognition of this form of respiration consists in the mixture of the vesicular and the tubular quality during inspiration. It is suggestive, and denotes incomplete consolidation. The more pronounced the solidification, the greater the bronchial element, and the nearer the affected area is to the surface, the greater the bronchial type of respiration. This form of breathing is heard during the stage of resolution in croupous pneumonia. It is a valuable sign in incipient pulmonary tuberculosis, in interstitial pneumonia, in hemorrhagic infarct, and in compression of the lung from tumors, fluid, or air. It must be borne in mind that occasionally the respiratory murmur over the right apex is less vesicular and higher in pitch than over the left—in other words, the right apex normally shows a mild type of harsh respiration or bronchovesicular breathing. Sometimes bronchial breathing has a metallic or even a musical tone, or may have the character of an echo. This may be imitated by breathing into a bottle or decanter, and has been termed *amphoric breathing*. It shows that air has entered into a cavity and that it has not been expelled with expiration. Amphoric respiration may be heard with inspiration, with expiration, or with both. When it occurs in but one respiratory act, it is more frequently heard in expiration alone, being louder and more distinct. This sound is present only in large pulmonary cavities and in pneumothorax. Amphoric respiration occurs in a pulmonary cavity with rigid walls. The cavity must be sufficiently large for a free communication with a fair-sized bronchial tube, and be situated close to the surface of the lung. Cavernous respiration has already been partly described in a previous section, under bronchial breathing. (See p. 71.) The term cavernous respiration is not accepted by the Germans. Simply defined, it is a variety of bronchial breathing. It has the character of respiration heard over a cavity. The inspiration is blowing, and the pitch somewhat lower than that in bronchial breathing. Expiration and inspiration have almost the same quality, duration and intensity being variable. *Bronchocavernous respiration* is a rare physical sign, as is also *vesiculocavernous*

respiration. The latter occurs where the cavity is surrounded by apparently healthy tissue.

Rales.—Rales are adventitious new sounds, occurring during the act of respiration, created in the bronchi, lungs, or pleura. When heard in the lungs or bronchi, they are termed *rales;* and when heard in the pleura, they are known as *friction sounds.* Broadly speaking, they are divided into two classes—*dry* and *moist* rales.

Dry Rales.—Dry rales are appreciated as piping or whispering and snoring or humming, being either high pitched or low pitched. The former are termed *sibilant rales;* and the latter, *sonorous rales.* They are produced by a diminution in the lumen of the bronchial tube, either through the deposit of a tough tenacious exudate, due to inflammation, or from pressure upon the tube by tumors, gas, or fluid. Sonorous rales are produced in the large, and sibilant rales in the small, bronchial tubes. They are heard both in inspiration and in expiration. Should they appear only in one of these two conditions, they are most likely to be heard in inspiration alone. The sonorous rales may be mistaken for pleural friction, but the differential diagnosis between the two conditions is not difficult, as the friction sound is limited in extent, whereas the sonorous rale is heard over a large area. These rales occur commonly in the first stage of acute bronchitis, in asthma, and in chronic bronchitis. Coughing may either increase or diminish the rale.

Moist Rales.—Moist rales are divided into large and into small mucous rales, into crepitant and into subcrepitant rales. Large moist rales are produced in the larger bronchial tubes and in cavities. They are caused by the passage of air through secretion, the current of air throwing the fluid into vibration in the act of respiration. Talma has given another explanation: "When we blow through a tube one end of which is immersed in water, it is supposed that the current of air separately moves the air-bubbles which present projections into the bronchial tubes, and that as one such quantity of air breaks the bridge through the fluid and advances, the fluid behind it immediately rushes on again and occupies the space and shares the vibrations in the pent-up air." Traube thought that the to-and-fro motion of the secretion produced by the current of air caused moist rales. The explanation most generally accepted to-day is the following: If air enters a bronchial tube in which secretion is present, the inspired

column of air forces the fluid down through the tube so tha at some level it causes the formation of a diaphragm, thi is pushed further and further through the tube until th lumen of the vessel becomes too narrow to hold it, when i breaks, producing a sound (the rale); the reverse take place in expiration: the diaphragm is again formed at th narrowest part of the tube, forced upward through the bron chus until the lumen of the tube is too wide to hold th fluid diaphragm, when it again bursts, and produces a soun recognized as a rale. This explains why moist rales are hear both in inspiration and expiration, with the single exceptio of the crepitant rale, which has another mechanism that wil be considered later on. The small mucous rale is produce in the same way as the large mucous rale. The larger an medium-sized moist rales are heard in the second stage of bronchitis, in the stage of asthma in which expectoration appears, in the second stage of acute bronchitis when fluid begins to form, and, in fact, in any condition in which fluid is present in the larger or medium-sized bronchial tubes. They are also heard over a cavity when fluid is present, and produce a sound of a gurgling character; hence, they are called *gurgling rales*. Occasionally, rales are heard which have a decided metallic quality. They occur over superficially situated cavities with smooth walls. This has been called *metallic tinkling*, and sometimes resembles the sound of falling drops. This rale is heard particularly in hydropneumothorax, and has also been called *gutta cadens*. Baas called attention to the fact that if fluid form at the top of a cavity, the drop does not fall directly to the lowest level, but gravitates along the walls; thus, the name gutta cadens, which was given to it by Laennec, describes the sound more than the actual condition.

The Subcrepitant Rale.—The subcrepitant rale is formed in the small bronchioles. It is heard in inspiration, in expiration, or in both. The element of moisture is pronounced, and gives the rale known as *crackling*. In occurs in congestion and edema of the lungs, in hemorrhage, or in any condition in which fluid is present in the finer air-passages; hence, the rales are occasionally present in the early stages of chronic pulmonary tuberculosis, and in the stage of resolution of croupous pneumonia.

The Crepitant Rale (*Crepitation*).—This rale has a peculiar acoustic property, which may be imitated by rubbing the hair

in front of the ear between the fingers. It is produced only in the alveolar structure of the lung, and is heard only at the end of a forced inspiration. It is due to the alveolar structure being collapsed and glued together, or when the alveoli are partially filled with secretion. Thus, it may occur in persons who have been bed-ridden in whom atelectasis of the lungs at the bases has appeared. After a prolonged inspiration, the alveoli separate as the air enters, and the rale is heard at the end of inspiration. It most commonly occurs pathologically from accumulation of fluid in the alveoli and parts of the fine bronchi. The rale appears commonly in croupous pneumonia, bronchopneumonia, hemorrhage, pulmonary infarcts, edema of the lung, and occasionally in pulmonary tuberculosis. Penzoldt has called attention to the fact that sometimes crackling or crepitation may be heard in expiration. This view has not received general acceptance.

Pleuritic Friction Sounds.—These are heard when the pleural surfaces are roughened, the intensity varying from the slightest rubbing to a shuffling or creaking sound, which may not only be heard, but may be readily transmitted to the hand through palpation. The sound may be heard either in inspiration, in expiration, or in both. It is close to the ear, superficial, and is increased by pressure with the stethoscope, which causes pain. The pronounced loud sound may be broken or interrupted during respiration. The low, less intense sounds are usually continuous. The sound disappears with the formation of fluid in the pleural cavity. It reappears as absorption takes place. Pleuritic friction is usually localized (circumscribed). Occasionally, a friction sound is heard that is synchronous with the heart-beat. This is known as *pleuropericardial friction*, and will be described under Pericardial Friction Sound. (See p. 91.) If the pleural friction sound occur in inspiration, it may be mistaken for the crepitant rale. The points of difference are these: The crepitant rale is usually not so circumscribed, being heard over a wider area; it accompanies consolidation, and is not influenced by cough, pain not being a prominent symptom.

Auscultation of the Voice (Vocal Resonance).—For diagnostic purposes the ear may be placed upon the chest (immediate auscultation), or the stethoscope may be used (mediate auscultation), and the patient asked to speak with the loud and with the whispered voice. If the stethoscope is placed over the larynx during the act of speaking, a loud, almost disa-

greeable, sound is transmitted to the ear. The voice is carried from the trachea into the bronchial tree. The vocal resonance will be greater under normal conditions, depending upon the size and the superficial situation of the bronchi. The development of the chest also influences the vocal resonance; thus, the more developed the chest and the greater the thickness of the muscular walls, the less pronounced the vocal resonance appears. Vocal resonance is less marked in children and in women than in men, and is more distinct in the aged than in middle life. In general terms it may be stated that vocal resonance and vocal fremitus are influenced by the same conditions. A possible exception may take place in the accumulation of fluids in the pleural cavity. Occasionally, although rarely, large pleural effusions may exist over which the voice will be transmitted to the ear. An increase of vocal fremitus is noted in all conditions of consolidation; hence it occurs in pneumonia and tubercular disease of the lungs. When the vocal resonance over the chest assumes the character that it has over the trachea, it is spoken of as *bronchophony*. This sometimes occurs over cavities, and becomes especially plain when the whispered voice is used, being then known as *whispering pectoriloquy*. Occasionally, a nasal bleating sound is heard, especially over pleuritic exudates. This condition is called *ægophony*. It is supposed to resemble the bleating of a goat; hence the name.

The Bacelli Sign.—Bacelli has described a sign to determine the nature of pleural exudates. According to Bacelli, the whispered voice will be heard through serous effusions, but not through purulent effusions. The sign is not reliable, and clinically very little importance should be attached to it.

The **amphoric voice** may be associated with amphoric respiration. It may occur either with a loud voice or a whisper; it is usually, however, more marked with the latter.

Bell Tympany.—This occurs when air is confined in the pleura. If the ear is placed over the chest anteriorly and two coins are tapped one against the other, at the opposite side of the chest, a distinct, metallic, ringing sound is transmitted to the ear. It occurs particularly in pneumothorax, never occurring over normal lung.

Succussion.—The succussion sound was first described by Hippocrates; hence it is termed *Hippocratic succussion*. It occurs only in conditions in which fluid and air are present in the pleural cavity. It is elicited by placing the ear upon the

chest and shaking the patient vigorously, when a splashing sound is transmitted to the ear. This sound is often apparent to the patient, and is observed with a change in position. The sound is exactly similar to that produced by shaking a large vessel partially filled by fluid. It takes place particularly in hydropneumothorax, and occasionally may be heard over the stomach when that organ contains gas and fluid.

PHYSICAL DIAGNOSIS OF THE HEART.

The methods of physical diagnosis employed in the examination of the heart consist in inspection, palpation, percussion, and auscultation.

Inspection of the Heart.—Inspection of the heart should be undertaken in a good light, preferably with the patient in the sitting or dorsal position. Daylight is the best for this purpose. The following points should be noted, and are of importance: *The apex-beat, its diffusibility*, and any *abnormal pulsation* in the cardiac area. The heart is inclosed in the pericardial sac and is placed obliquely in the chest. "The broad attached end or base is directed upward and backward to the right, and corresponds to the interval between the fifth and eighth dorsal vertebræ. The apex is directed forward and to the left, and corresponds to the interspace between the cartilages of the fifth and sixth ribs in the parasternal line. The heart is placed behind the lower two-thirds of the sternum, and projects further into the left than into the right cavity of the chest, extending from the median line about three inches in the former direction, and only one and one-half inches in the latter. Its upper border would correspond to a line drawn across the sternum on a level with the upper border of the third costal cartilages, and its lower border to a line drawn across the lower end of the gladiolus from the costoxiphoid articulation of the right side to the point previously mentioned as the situation of the apex. The lungs cover a large part of the heart, and during inspiration when their borders meet behind the sternum a thin layer of lung covers the roots of all the large vessels; hence, the custom of making the patient hold the breath while examining the sounds of the heart" (Gray).

The Apex-beat of the Heart.—If the chest of a healthy individual is examined, it will be noted that in the fifth left intercostal space, at or near the parasternal line, there is a circum-

scribed rhythmic bulging, which is known as the apex-beat of the heart. It occurs with the systole or first sound of the heart, and is almost coincident with the pulse of the carotid and radial arteries; more correctly stated, it is perhaps slightly in advance—0.0093 of a second before the carotid artery and 0.0224 of a second before the radial artery. The breadth of the apex is about 2.5 cm., showing that it can be easily covered with the tip of one finger. In early childhood the apex-beat is often situated higher: in the fourth year it is most often found in the fourth intercostal space. The apex-beat of the heart is not visible in all persons. In some few individuals it is often not perceptible to the eye; occasionally, in women who have a short thorax and small intercostal spaces, the apex-beat may not be noted. In all such cases palpation must be resorted to. The thinner the chest-wall, the plainer the apex-beat; hence, it is usually noted to a greater extent in children than in adults. Several points are of importance in consideration of the apex-beat: the *position*, the *breadth*, the *strength*, the *time*, and the *rhythm*.

Position of the Apex-beat.—As has already been indicated, the apex-beat in children is situated somewhat higher than in adults, whereas in the aged in may be as low as the sixth intercostal space. In deep inspiration the apex-beat may be displaced downward one or more intercostal spaces, returning, however, to its normal place with deep expiration. It is more apparent in expiration, approaching nearer the mammillary line at this time. On the other hand, it may partly or completely disappear on inspiration, as the overlapping left lung may entirely cover it. The position of the apex-beat changes with posture. It falls from three to six centimeters to the left, and from one and a half to three centimeters to the right, accordingly as the body lies upon the left or upon the right side. Slight retraction also takes place if the sitting or upright posture is exchanged for the horizontal. Bodily exercise and mental emotion have some influence upon the apex-beat. It may become stronger, broader, and change its position a little more to the left and downward under these influences. Congenital deformities have some effect upon the position of the apex-beat of the heart; thus, in dextrocardia the heart is found upon the right side, in the same position that it should normally assume upon the left side. It will also be found that other organs have changed their position; thus, the liver will be found upon the left side, the

spleen upon the right, the cardiac end of the stomach upon the right side, and the pylorus upon the left side, etc. Other malformations have been described in the chapter upon Diseases of the Heart. (See p. 313.) Alterations in the contour of the heart produce changes in the position of the apex. Among the diseases of the lungs chronic pulmonary emphysema and interstitial disease produce dislocations of the apex-beat. In pulmonary emphysema the volume of the lung is increased so that the diaphragm, and with it the heart, are pushed down lower than their normal positions. In interstitial pneumonia the heart, on the contrary, is drawn in the opposite direction. Great changes take place in the position of the apex-beat from disease of the pleura. Accumulations of gas or fluid in the pleural cavity push the apex-beat to the opposite side. In diseases of the right pleura the apex-beat may even be found in the left axillary line, whereas in left-sided disease the change is not apt to be so great. Friedreich has called attention to the fact that in right-sided pleural effusion the apex-beat is also pushed forward. It not infrequently happens that from disease of the pleura the apex-beat may permanently assume the position that it first received in its dislocation. Diseases of the mediastinum, such as enlarged lymphatic glands, displace the apex-beat downward and outward. Diseases of the abdominal organs, tumors, and accumulations of gas or fluid in the peritoneum cause a dislocation of the apex-beat upward and outward. Gerhardt has called attention to the fact that dislocation of the apex-beat does not occur in pregnancy. Further, it must be mentioned that the apex-beat may be displaced from disease of the circulatory apparatus itself: thus, a downward displacement occurs from aneurysm of the aorta; a downward displacement is not infrequently noted from accumulation of fluid in the pericardium. Finally, diseases of the heart-muscle produce marked changes in the position of the apex-beat; thus, from disease of the left ventricle the apex-beat is often displaced downward to the left, showing hypertrophy.

Breadth of the Apex-beat.—The breadth of the apex-beat varies considerably even under normal circumstances. Exercise and emotion may cause an increase in the breadth of the apex-beat. It also becomes broader in expiration in the upright position and in a forward leaning of the body, so that the heart approaches nearer the thorax wall. Pathologic conditions show an increase in the breadth of the impulse in all

cases in which the heart comes nearer the thorax, especially if associated with retraction of the left anterior border of the lung. It results particularly from increase in substance of the left ventricle.

Force of the Apex-beat.—This is estimated by the degree of resistance which the heart offers to the finger when placed over the position of the impulse. A strong impulse is called *heaving* and *resistant*. To appreciate these differences, the student must be familiar with the force of the normal apex-beat. A powerful, heaving, resistant apex-beat is a sign of hypertrophy of the left ventricle. As a rule, all conditions that produce a slowing of the cardiac action are combined with increased force of the apex-beat. Occasionally, this may be due to febrile conditions, and in those cases of palpitation from which nervous and hysteric persons are apt to suffer. An enfeebled impulse is due to the loss of power in the heart muscle, and is an important sign in myocardial disease. A weak or absent impulse may be due to a portion of lung intervening between the heart and the thorax, and is very likely to occur in chronic pulmonary emphysema. A like condition is apt to take place from an accumulation of fluid in the pericardial sac. Under normal circumstances in deep inspiration the impulse becomes weaker for the reason that the left lung at that time will be found to cover a larger part of the cardiac area. In pathologic conditions this fact is of importance. Thus, it occasionally occurs from extrapericardial adhesions that the impulse may become more powerful and apparent during full inspiration. The impulse may be weakened or entirely absent from adhesion of the two layers of the pericardium. Finally, changes in the wall of the thorax itself may produce a weakened or absent impulse, such as edema of the chest-wall, inflammatory conditions, and a large accumulation of fat.

Systolic Retraction.—Systolic retraction is encountered in the region of the apex-beat, in which several interspaces of the thorax wall are drawn in with every systole of the ventricle. With diastole the retracted parts again come forward and almost give the appearance of a diastolic apex-beat. The retraction is usually more apparent in inspiration than in expiration. This sign is noted in adhesive pericarditis, especially in the form known as mediastinal pericarditis. The sign is also known as *systolic dimpling*. Simpson has called attention to the fact that the retraction only occurs at the latter

part of the systole, as may be observed by keeping the finger over the carotid or radial arteries.

Rhythm of the Apex-beat.—As a rule, the apex-beat is felt as a systolic impulse; occasionally, however, a double impulse is felt. This doubling of the impulse, according to Leyden, is due to a separate contraction of the right and left ventricles. These contractions of the ventricles occur independently of each other, so that there is practically a hemisystole. This condition has been called a *reduplicated first sound.* The production of the apex-beat of the heart is by no means settled; several theories have been advanced as to its production: Bamberger thought that it concerned the apex of the heart alone; the theory generally accepted is that the heart-beat occurs about in the middle of the time between the closing of the auriculoventricular and arterial valves, that it takes place in the first part of the systole, which has been called the time of closing (Verschlusszeit). This is the time at which the ventricle is filled with blood, and in which all valves are still closed. It is then due to the change in the form of the heart muscle. These observations were made by Martin by means of the cardiograph; they are, however, by no means generally accepted.

Diffused Impulse.—As has already been suggested, normally the impulse of the heart is limited to the fifth intercostal space upon the left side in the parasternal line. Should the impulse be seen over a larger area, it is spoken of as a *diffused* impulse. This results very often from dilatation of the right ventricle, and may be seen to the right of the left sternal line and even in the epigastric region. If hypertrophy should coexist, the impulse becomes more powerful.

Prominence or bulging in the cardiac area, which may include both the sternum and the ribs, may take place from hypertrophy and from dilatation. Pericarditis with effusion also gives rise to distinct bulging. Pulsations are sometimes localized,—they may be limited to the base of the heart in the second intercostal space on the right and left sides,—and may be due either to the aorta or the pulmonary artery. If these pulsations are systolic in time, they are likely to indicate aneurysm of these vessels. From what has been said under inspection it will be noted that the lowest tip of the heart, the actual apex (anatomic apex), rarely comes against the chest-wall, that the systole of the heart that can be seen and felt is due to the right ventricle, so that, clinically at least, the right

ventricle is the apex of the heart, whereas the left ventricle forms the anatomic apex.

Palpation of the Heart.—Palpable Apex-beat.—The apex-beat is felt as a short, sharp shock, which is systolic in time. It has no particular diagnostic significance. Frequently, a diastolic shock of moderate intensity may be discerned at the base of the heart, which is also normal. The diastolic shock (which is normal) is found to be diffused; it assumes diagnostic importance when it becomes localized. This occurs most often in the second left intercostal space close to the sternum, in the region of the semilunar valves of the pulmonary artery. The reason for this may be due to favorable circulatory conditions or to increased force of the pulmonary valves. Favorable circulatory phenomena occur when the border of the left lung that covers the pulmonary artery has become infiltrated and airless or is retracted so that the pulmonary artery lies closer to the chest-wall. Under such circumstances, visible pulsation over the pulmonary artery may be noted. Increased force of the pulmonary valves takes place when difficulty or great resistance is encountered in the right ventricle. This occurs most frequently from disease of the mitral valve and in chronic pulmonary affections. In the two right intercostal spaces near the sternal border the diastolic shock is rarely encountered. It occasionally results from hypertrophy of the left ventricle, due to contracted kidney.

Palpable Murmur.—Pathologic changes in the heart frequently give rise to adventitious sounds. These are particularly noted upon auscultation, but may sometimes be discernible by palpation. Depending upon the position in which they arise and their character they are known as *endocardial* or *pericardial* sounds. Ordinarily, pericardial and endocardial sounds can be differentiated by the sensations which they impart to the finger. Pericardial murmurs are known as *friction sounds.* They give the hand the sensation of rubbing, scratching, or shoving, and are characterized by marked interruptions, whereas endocardial sounds that are apparent to the finger have a more continuous character, much like the purring of a cat or the sound of a violin string which is thrown into vibrations. The differential diagnosis is rendered easier if the sound is only discernible upon strong pressure in one interspace. This would favor the fact that the sound was of pericardial origin, being produced through the closer proximity of the pericardial layers upon pressure. It is also

important to notice the time at which these phenomena occur.

Endocardial sounds have a definite relation to the phases of the action of the heart, and are systolic, diastolic, or presystolic in time. Pericardial friction sounds differ markedly from this: they are neither strictly systolic nor diastolic, but may occur independently of systole or diastole, and are not definitely related to either the first or second sound. If there should be still further doubt, auscultation should be resorted to. Both murmur and friction rub may disappear in deep inspiration to the palpating finger. According to the sensations that the endocardial murmur give to the finger, they are known as purring and frèmissement cataire (Laennec). The sound is also described as a thrill. On the other hand, the palpable pericardial friction sounds are spoken of as friction rubs, usually giving rise to a loud sound to the ear. They may partially disappear when the heart is acting quietly, but are always increased by exercise or mental emotion, or by a change from the dorsal decubitus to the upright posture. The thrill is of great importance in the diagnosis of valvular disease of the heart. When present, it is a most important sign of stenosis of the valve according to the position in which it occurs. A thrill at the apex, presystolic in time, is significant of mitral stenosis. A thrill in the second right intercostal space, systolic in time, is diagnostic of aortic stenosis. Thrills occurring in relation to the right heart are exceedingly rare, but a thrill in the region of the tricuspid valve, presystolic in time, would point to tricuspid stenosis, and a thrill in the second or third interspace upon the left side at the base would be suspicious of stenosis of the pulmonary valves. The presystolic thrills are further characterized in that they are usually more marked at the beginning and end of the thrill than during its middle course. Palpable pericardial friction sounds only occur when inflammatory changes take place in the pericardium. Pericardial friction sounds are most frequently felt in the neighborhood of the left sternal border.

A rare sign elicited upon palpation occurs over the trachea in some cases of aortic aneurysm. The observer should stand behind the patient, and his fingers should firmly grasp the cricoid cartilage. The patient should rest the chin upon the chest, and he should be told to hold the breath. An upward and downward motion of the trachea is noted if the sign is present. It is known as *tracheal tugging*.

Percussion of the Heart.—Percussion of the heart should be practised by using the fingers. The patient should be in the recumbent posture or slightly elevated, assuming a partial sitting position. But a small portion of the heart is uncovered by lung. This extends from the fourth to the sixth costal cartilages. The area of superficial dullness does not extend more than two inches in any direction. It has a triangular shape, the apex being below the juncture of the third left rib with the sternum, and the base being on a line with the cartilage of the sixth rib. Normally, the area of deep-seated dullness extends from the left nipple to about one-half of an inch to the right of the sternum transversely, and from the second to the sixth interspaces vertically. The lower border of the heart belongs to the right ventricle; the left border consists of the left ventricle, beginning about at the middle of the second left intercostal space and extending downward to the fifth left intercostal space. Percussion of the heart, as will be noted from the description, is exceedingly difficult, and can only be practised with some degree of accuracy in cases in which pericardial effusions take place. Recognizable changes upon percussion of the heart occur if gas or fluid is present in the pericardial sac. In the case of gas a tympanitic note is found where cardiac dullness should be encountered; if there be fluid, great changes take place in the pericardium. Considerable increase in the size of cardiac dullness and absolute flatness upon percussion are noted. In very large effusions the greatest area of flatness is noted at the apex of the heart, merging into dullness as the normal base of the heart is reached. The shape of the flatness is described as being triangular, with the base of the triangle toward the apex of the heart.

Auscultation of the Heart.—Auscultation of the heart should be practised by means of the stethoscope, each valve position being carefully auscultated. It is apparent to any one familiar with the anatomy of the heart that the area of a silver dollar pressed upon the heart would come in contact with each of the four valves; hence, it has been necessary to study carefully the points at which the valve sounds could be individually determined. The point of maximum intensity at which the valve sound can be heard is called the *punctum maximum*. Anatomically, the position of the mitral valve is about at the third left costal cartilage, near the sternum. The position at which it should be auscultated is at the apex of the heart.

The situation of the tricuspid valve anatomically is in a line drawn between the third left intercostal space and the fifth right costal cartilage. The punctum maximum is in the median line at about the fifth right costal cartilage. The anatomic location of the aortic valve is between the median line and the third left costal cartilage. The punctum maximum is the second right costal cartilage, sometimes called the *aortic cartilage*. The anatomic position of the pulmonary valve is in the middle of the second left costal cartilage, 1.5 cm. to the left of the sternal border. The punctum maximum is between

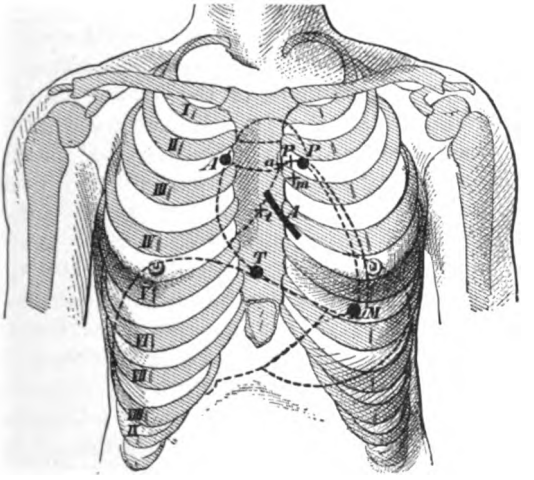

Fig. 7.—The anatomic situation and the points for auscultating the valves of the heart and its orifices. The crosses indicate the anatomic situation; the black points and lines indicate the places to auscultate. The small letters show the location of the valves; the large ones, the points for auscultating: *a A*, The aorta; *m M*, mitral valve; *p P*, the pulmonary orifice; *t T*, tricuspid (Vierordt).

the second and third left intercostal spaces, near the left sternal border. In auscultation it is necessary to differentiate between the sounds of the heart and murmurs that may be present, and a further distinction must be made between endocardial and exocardial murmurs. The exocardial murmurs occur in disease of the pericardium, and are identical with friction sounds.

Auscultation of the Heart-sounds.—If the ear is placed over the cardiac area in a normal individual, two distinct sounds are encountered—one is called the systole or first sound, the other the diastole or second sound. They are

separated from each other by a short pause, whereas a longer pause occurs between the second and first sounds. The first sound is due to the contraction of the ventricle, producing the muscular element, the rush of the blood, and the closure of the auriculoventricular valves; the second sound is due to the closure of the semilunar valves in the aorta and pulmonary artery. The first sound is then synchronous with the apex-beat, and is the long sound; the second is the short, sharp, valvular sound. The first sound is heard with greatest intensity at the apex of the heart; the second sound is best studied at the base.

Rhythm of the Heart-sounds.—The first sound is characterized by a dull, deep, more prolonged, and less sharply defined hum than the second higher pitched, snappy, valvular, diastolic sound. The strength of the cardiac sounds depends upon the thickness of the thorax. In a thin thorax the sounds are more pronounced than in a thick-walled chest. Edema of the chest-wall may show a decided influence upon the intensity of the heart-sounds. In deep inspiration the strength of the cardiac sounds diminishes, as is also the case in decided pulmonary emphysema. In consolidation of the lung, on the other hand, this acts as a favorable medium for the transmission of heart-sounds, as do also cavities in the lung. In syncope the heart-sounds may be so feeble that they can not be heard. The same is true, especially of the first sound, in many of the infectious diseases, in fatty infiltration of the heart-muscle, and in many forms of myocarditis. The second sound of the heart is accentuated in all conditions in which an increase of the cardiac muscle takes place, especially in hypertrophy of the left ventricle. The same is true in arteriosclerosis and in contracted kidney. Accentuation of the second pulmonary sound is an important sign of hypertrophy of the right ventricle. An accentuation of the systolic sound takes place at the apex, as was first pointed out by Traube in mitral stenosis. The reason lies in the fact that from stenosis of the auriculoventricular opening of the left side of the heart the blood can only slowly enter the ventricle, so that as less blood is present in the ventricle the contraction of the ventricle becomes more powerful, showing this by accentuation of the first sound. A diminution in the intensity of the first sound may occur from aortic insufficiency. It is due to the fact that at the end of diastole the mitral valve has already become tense, and so the increase of the tensity of the valve

during systole, in final closure, is not well marked. Diminution in the intensity of the second sound at the aorta and pulmonary artery may be due to diminished blood-pressure and from the reduction in the contractile power of the semilunar valves from thickening. The second sound may be entirely absent, especially the aortic, in mitral regurgitation and stenosis. In a high grade of stenosis of the mitral valve very little blood enters from the auricle into the ventricle, so that at the following systole a very small amount of blood is sent from the ventricle into the aorta, causing the aortic valves to close with but slight force, the second sound being weakened or entirely absent. The second aortic sound is often accentuated, and sometimes has a ringing quality. In arteriosclerosis a metallic sound is sometimes added, also if large cavities are situated in the lungs near the heart. This may result from tubercular disease of the lungs, pneumothorax, pneumopericardium, and occasionally from meteorism. It has also been observed in adhesive pericarditis.

Reduplication of the Heart-sounds.—Occasionally, there is a reduplication of the heart-sounds in which the rhythm of the heart is altered, depending upon whether the reduplication be systolic or diastolic. Should it occur with the systole, it is particularly noted at the apex or at the punctum maximum of the tricuspid valve. If it takes place with the diastole, it is most likely to be heard at the base, at either the pulmonary or aortic orifices. The cause of reduplication of heart-sounds is due to the failure of synchronous action of the valves or the ventricles. The condition occurs commonly in valvular disease from thickening or retraction of the valve segments, and in some cases of disturbance of innervation, as when the papillary muscles contract at different times.

Under the name of *gallop rhythm*, a peculiar reduplication of heart-sounds has been described, in which the accentuation is laid upon the middle tone, so that the sound is not unlike the galloping of a horse. There is no satisfactory explanation of this condition. It has been found in emphysema with circulatory disturbance, in arteriosclerosis, and in cases of insufficiency of the mitral valve. According to Fraentzel, the gallop rhythm points to grave cardiac weakness, and is an unfavorable prognostic sign. It is encountered in the severe infections, in the cachexia of malignant diseases, and in severe anemic conditions. Friedreich has called attention to the reduplication of the second sound occurring in pericarditis, in

which the layers of the pericardium have fused together, accompanying systolic contraction.

Auscultation of Endocardial Murmurs.—We owe this important physical sign almost entirely to the researches of Skoda, who called attention to the fact that in the auscultation of murmurs two things were of particular importance—the position in which the murmur occurred with the greatest intensity, and its time in the cardiac revolution. The situation in which the murmur is heard with greatest intensity almost invariably points to the orifice at which the diseased valves are situated, while the time at which the murmur occurs points to either regurgitation or stenosis, or both. The acoustic character of the individual murmur is without diagnostic importance. All degrees of variations of sounds are heard. The murmurs have been described as rasping, sawing, snoring, blowing, whistling, and so on. Occasionally, a high-pitched piping, or even singing, quality may be noted, when the term *musical murmur* has been used to signify the condition. In the vicinity of large cavities murmurs may even have a metallic quality. The intensity of the murmur also shows great variability. The question of muscular force of the heart very largely enters into this. If the heart be acting quietly, murmurs may disappear entirely, and only reappear when the heart's action is most active—as a result of bodily exercise or mental emotion. In many cases posture is of great importance ; as a rule, in the erect posture endocardial murmurs are less intense or may disappear altogether ; rarely, the opposite of this may occur. The reasons for these phenomena are not clear, however, the lesson to be learned from them being that if valvular disease be suspected, the patient should be examined in several postures. Occasionally, murmurs may be heard at some distance from the chest. As a rule, they are the murmurs of stenosis, particularly of the aortic valve. The rule that the position of the greatest intensity of the endocardial murmur shows the valve that is affected, may generally be adhered to ; there are, however, slight exceptions to this : for instance, the murmur of aortic insufficiency is often heard with greater intensity at the ensiform cartilage than at the second right cartilage, as the murmur does not have its origin in the beginning of the aorta, but in the left ventricle. Occasionally, the murmur of mitral regurgitation may be heard with greater intensity at the pulmonary area than at the apex of the heart. In reference to the time that

endocardial murmurs arise they are divided into systolic and diastolic murmurs; however, there is a murmur that occurs just before systole, known as the presystolic murmur, which is of great importance in the diagnosis of mitral stenosis. We then may divide organic murmurs into systolic, presystolic, and diastolic murmurs. Endocardial murmurs are divided into *organic* and *functional* murmurs. Organic murmurs are due to some anatomic alteration of the heart-muscle or of the valves. The functional murmurs occur in febrile conditions and in anemic affections, or diseases in which profound alterations of the blood take place; hence, they are known as *hemic*, *functional*, and *inorganic* blood murmurs. The organic murmur is produced by fluid veins occurring in the blood stream. Physically, these fluid veins must occur in all cases in which the blood is forced suddenly to enter from a narrow opening into a wide one, or where two blood streams coming from opposite directions meet. This is known as the "*Chauveau's fluid vein theory.*" It accounts for the production of the organic murmur, but not for the functional murmur. No causes have been given that will satisfactorily explain the production of the functional or hemic murmur.

Stenosis of the Aortic Orifice.—With the contraction of the ventricle, under normal conditions, the aortic valves should be open. If narrowing occurs, with the contraction of the ventricle, a systolic murmur is produced at the aortic cartilage due to the blood stream being impeded by the narrowed valve. This murmur often shows great intensity, and not infrequently has a musical character. This systolic murmur is transmitted to the vessels of the neck (carotid artery).

In **aortic regurgitation** a diastolic murmur is produced at the aortic cartilage, due to the insufficient closure of the semilunar valve, allowing the blood to regurgitate into the left ventricle. This murmur is often heard with greater intensity at the ensiform cartilage than at the aortic cartilage.

Mitral stenosis shows itself by a presystolic murmur, which is heard with greatest intensity at or near the apex. With the diastole of the heart the blood is forced from the left auricle through the narrowed mitral valve, so that the murmur must take place before the contraction of the ventricle, which is the systole. This murmur is not transmitted and is accompanied by a presystolic thrill.

Mitral regurgitation is diagnosticated by the presence of a systolic murmur at the apex, as with the contraction of the

ventricle blood is forced through the mitral valve back into the left auricle on account of the insufficient closure of the mitral valve. This murmur is transmitted into the axilla and to the angle of the scapula.

Diseases of the right heart are extremely rare; when they take place, the pulmonary valve lesions show similar murmurs to the aortic. The tricuspid lesions and the mitral lesions show the same murmurs. It must furthermore be added that the valve lesions characterized by stenosis almost always are accompanied by a thrill that has the same rhythm as the murmur. Functional murmurs are always systolic in time. They are heard most frequently at the bases of the heart, particularly at the left base. They are not transmitted. Occasionally, the sound is heard in the veins of the neck, known as the venous hum.

The differential diagnosis between the organic and functional murmurs is, as a rule, not difficult.

	Organic Murmur.	*Inorganic or Functional Murmur.*
Time	The time may be systolic, diastolic, or presystolic.	Always systolic in time.
Position	Heard at the punctum maximum.	Heard at the base of the heart, particularly at the left base.
Transmission	Transmitted, except the presystolic mitral murmur.	Never transmitted; occasionally heard in the neck.
Changes in the heart-muscle	Changes in the heart-muscle always occur. Hypertrophy or dilatation or both.	Changes in the heart-muscle do not occur.
Character of the murmur	May be harsh or musical.	Always soft and blowing in character.

Auscultation of Exocardial Murmurs or Pericardial Friction Sounds.—Exocardial or pericardial murmurs are friction sounds. They may often be diagnosticated by their acoustic character. They are coarser and have a grazing, scratching, rubbing sound, or even a sound resembling the creaking of new leather. Under rare circumstances pericardial friction sounds may be soft, when great difficulty will arise in the differential diagnosis between endocardial and exocardial sounds.

The differential diagnosis must be made in the following manner:

Endocardial Murmur.	*Pericardial Friction Sound.*
The endocardial murmur is heard at the punctum maximum. It is distinctly presystolic, systolic, or diastolic.	The pericardial friction sounds do not conform strictly to these phases; they may occur between, and may overlap, systole or diastole.

Endocardial Murmur.	*Pericardial Friction Sound.*
Are often obliterated by pressure with the stethoscope.	Pressure with the stethoscope often intensifies the pericardial sound.
Are often decreased through deep inspiration.	Are increased in intensity during deep inspiration.
Are transmitted.	Are localized to the cardiac area, and have the appearance of being immediately under the ear.
Pain usually absent.	Pain present.

The cause of pericardial friction is most frequently due to inflammation of the pericardium. The intensity of the friction sound varies greatly. It may be so loud that it is apparent to the patient; on the other hand, it may be weak or entirely absent.

Occasionally, a condition has been noted which has been called *pleuropericardial* friction. This may occur when the pleura of the left anterior border of the lung lying nearest the heart is inflamed so that the pericardium shares in the process. This may be differentiated from true pericardial friction in the fact that the sound is directly related to inspiration and expiration, disappearing as the breath is held.

EXAMINATION OF THE ARTERIAL SYSTEM.

Visible Pulsation.—The visible expression of the activity of an artery consists in the rhythmic filling of the artery synchronously with the systole of the heart, known as pulsation. Under normal circumstances pulsation even in the large arteries is scarcely visible. Changes, however, occur if the activity of the heart is increased. Under these circumstances a rhythmic beat is noted in the neck, and even the small arteries, such as the temporal, may show visible pulsation. This occurs from prolonged muscular effort, from excitement, in febrile conditions, and in disturbance of innervation of the heart's action. Visible pulsation is particularly noticeable in hypertrophy of the left ventricle, especially in aortic insufficiency, which is characterized by great hypertrophy of the left ventricle.

Capillary Pulse.—This was first described by Quincke. Occasionally, in healthy individuals, alternate blushing and pallor under the nails may be noticed synchronously with the systole and diastole of the heart. This may also be noticed by briskly rubbing the skin over the forehead. The intensity of the capillary pulse is increased in insufficiency of the aortic

valves. It occasionally takes place in aneurysm of the aorta, and it has not infrequently been observed in chlorosis.

Epigastric Pulsation.—Epigastric pulsation is noted below the xiphoid cartilage. It may be due to the pulsation transmitted from the abdominal aorta, celiac axis, or be due to the heart-muscle itself. Pulsation is noted in this region when the heart is abnormally situated. This may occur from chronic pulmonary emphysema, from hypertrophy of the right ventricle, from left-sided pleurisy, or from pericarditis with effusion. Visible pulsation in the epigastric region not infrequently occurs in hysteric and nervous individuals. A pulsation visible in the epigastric region also takes place from aneurysm of the abdominal aorta.

Pulsation from Aneurysms.—Aneurysms of superficially situated arteries show themselves as pulsating tumors. In aneurysm of deep-seated arteries a tumor only becomes visible after the overlying parts have become indurated or moved aside. Care must be taken not to confound every pulsating tumor with aneurysms, as solid tumors may overlie an artery and receive transmitted pulsations; in such cases the diagnosis is made by palpation. In a tumor overlying an artery palpation simply reveals a rising and falling of the mass as the artery dilates and contracts; whereas in aneurysm an *expansile pulsation* is noted. Frequently, also, in the case of the aneurysm there is a systolic thrill transmitted to the finger.

Transmission of Murmurs in the Arterial System.—In auscultating the larger arteries, such as the subclavian or carotid, in health two sounds are noted that resemble the cardiac sounds. In auscultating the femoral artery only a single, dull, systolic, almost toneless sound is audible. Under morbid conditions murmurs may be heard in the carotid and subclavian arteries, as in anemia, occasionally in exophthalmic goiter, and in valvular disease of the heart, particularly aortic stenosis.

EXAMINATION OF THE ABDOMINAL ORGANS.

For purposes of description the abdomen is divided into nine regions by drawing two horizontal and two vertical lines over the surface. The higher horizontal line is drawn just below the costal border of the ribs, and the lower one between the anterior superior spine of the ilium. The two vertical

lines are drawn from the center of Poupart's ligament upward. The regions are thus divided into the right and left hypochondriac regions, the right and left lumbar regions, the right and left iliac regions, the epigastric region, the umbilical region, and the hypogastric region.

In the right hypochondrium the right lobe of the liver with the gall-bladder and the hepatic flexure of the colon are found. More deeply situated are two-thirds of the duodenum, and still more deeply situated is the top of the right kidney with the suprarenal capsule. In the left hypochondrium are found the spleen, the splenic flexure of the colon, and the cardiac extremity of the stomach ; and more deeply situated is the left kidney with its capsule. The lumbar regions contain the kidneys, portions of the small intestines, and the colon. The right iliac region contains the cecum ; and the left, the sigmoid flexure of the colon. The epigastric region contains the body of the stomach, with the pyloric end, and the left lobe of the liver. More deeply situated are the pancreas, celiac axis, the hepatic vessels, and the semilunar ganglia.

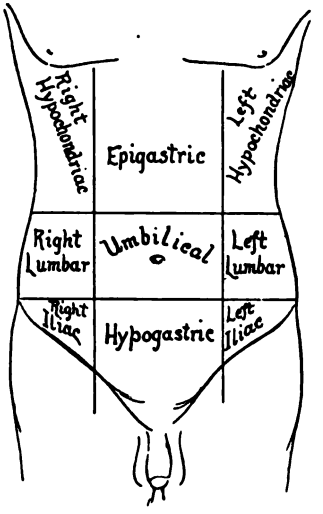

Fig. 8.—Lines drawn upon the surface of the abdomen, dividing it into regions.

The umbilical region contains in its upper part the transverse colon, and behind this the duodenum ; however, the greater part is made up by the small intestine. The hypogastric region contains the coils of the small intestine and the mesentery. The aorta divides a little to the left of the umbilicus, and a distended bladder or a pregnant uterus may rise into the hypogastric region.

Inspection of the Abdomen.—This is of great importance. It should be preferably undertaken with the patient first in the erect posture and then lying flat upon the back. The *size* and *shape* vary greatly in health in different individuals. A general enlargement may occur from subcutaneous fat of the abdominal walls or of the mesentery. It may be due to edema. Ascites causes a change in the shape of the abdomen, and it

will be noted that the shape of the abdomen changes with alterations of posture. A small quantity of fluid may only be indicated by slight bulging in the flanks. A large quantity of fluid increases the bulging in the flanks, but also causes a rounded top, and may cause the entire abdomen to become barrel shaped. Meteorism (distention of the bowel by gas) may cause extensive enlargement of the abdomen. Finally, enlargement of individual organs—such as the spleen, liver, stomach, and other organs—and morbid growths may produce the condition. Retraction or a diminution in volume of the abdomen may occur from general emaciation, in stricture of the esophagus or of the cardiac end of the stomach. Movements may occur in the abdominal region not only from respiration, but from peristalsis. Peristaltic movements are rarely visible in health. When present, they are usually caused by distention of the intestines or stomach. Pulsation may be due to aneurysm or to tumors and glands overlying the aorta.

Pulsating aorta is not uncommon in anemic and in nervous women. A dilated condition of the superficial veins of the abdomen shows engorgement of the portal system, due most often to disease of the terminal vessels of the portal vein, or from pressure upon the portal vein or the inferior vena cava.

Palpation of the Abdomen.—For the purpose of investigating diseases of the abdomen by palpation it is necessary that the patient should be in the dorsal decubitus, with the shoulders slightly raised and supported, the thighs flexed upon the abdomen, and the chin touching the sternum. Normally, the abdomen is soft, yielding readily to pressure. If it should prove unyielding, the administration of an anesthetic may be required in some instances. The physician's hand should be gently laid upon the abdomen, firm pressûre being exercised after a short interval. In this manner upon deep pressure the kidneys, aorta, and the vertebral column may be felt.

Resistance.—Resistance is usually due to some morbid condition beneath the parts palpated. It may be an important sign of a deep-seated lesion. Occasionally, the resistance is associated with swelling that may be due to muscular contraction. In percussion over these enlargements a dull note may be obtained. In the majority of instances this condition is termed "phantom tumor," and an absolute diagnosis may be made by the administration of an anesthetic, when the tumor will disappear. This occurs particularly in neurotic individuals.

Pain.—Diffused tenderness is most frequently due to inflammatory changes in the peritoneum. It may also take place as a manifestation of hysteria. Localized tenderness may result from an underlying morbid condition, such as appendicitis, gastric ulcer, abdominal aneurysm, etc.

Fluctuation.—If fluctuation is present, it is usually due to an excess of fluid in the peritoneum. It should be elicited in the following manner: One hand should be placed flat upon the side of the abdomen, the opposite side of the abdomen being tapped with the fingers of the other hand. If fluid is present, a wave or fluctuation is communicated to the other hand. This only occurs if the fluid is free in the peritoneal sac, and does not occur if the fluid is encysted, unless the cyst be very large. The sense of fluctuation is more marked the greater the amount of liquid.

Percussion of the Abdomen.—By percussion the condition of the abdominal organs may be determined. The presence of liquid or gas, the increased size of organs, and morbid growths may be ascertained by this method.

Fluid in the Abdomen.—Upon percussion a flat note is elicited over fluid; and as the fluid seeks the most dependent part, small amounts are detected above the pubis by a flat note upon percussion. Larger amounts of fluid produce flatness in the recumbent posture, even in the hypogastric region. If the fluid is not encysted and free in the abdominal cavity, change of position produces change in note; thus, if the patient is placed upon the left side, the previously present flat note upon the right side gives way to clearness or even tympany upon percussion. In the recumbent posture a tympanitic note, with large quantities of fluid, is present in the umbilical region, as the intestines are apt to float forward and be present in this position, the fluid having gravitated to the sides. If adhesions are present, the fluid may not gravitate with change in position, as the fluid may be shut off in this way from the rest of the abdominal cavity.

A distended bladder and a pregnant uterus give rise to flatness upon percussion in the suprapubic region.

Gas in the Abdomen.—When the abdomen is distended by gas, a uniform tympanitic note upon percussion over the entire abdomen is elicited. If the quantity of gas be excessive, it may obliterate the lower borders of liver dullness, and the spleen.

Ascites not associated with dropsy in other localities is

found in acute peritonitis, tubercular peritonitis, cancer of the peritoneum, colloid disease of the peritoneum, chronic peritonitis, atrophic cirrhosis of the liver, and cancer of the liver. Ascites associated with dropsy elsewhere is the result of tricuspid disease, following left-sided valvular disease, in chronic pulmonary emphysema, in diseases characterized by weakness of the cardiac muscle, in tumors pressing upon the inferior vena cava, and in renal disease.

Physical Examination of the Stomach.—Five-sixths of the stomach is found to the left, and only one-sixth of the body to the right, of the median line. The cardiac orifice is situated slightly to the left in front of the eleventh dorsal vertebra. The fundus is situated principally in the left hypochondriac region, and may be partly covered by lung, especially when it is distended with gas. The pyloric end is situated close to the gall-bladder. Inspection under normal circumstances shows nothing to indicate the situation of the stomach. If enlargement of the stomach takes place (gastrectasis), the stomach being filled with gas, the outlines of the organ may be seen. The method of inflating the stomach is often practised for diagnostic purposes; for this the patient is given an ordinary Seidlitz powder with very little fluid. Palpation reveals tender points, tumors, or enlarged glands in the region of the stomach. It is not possible to map out the size of the stomach by means of percussion. The position of the fundus of the stomach is of great importance in diagnosis. It is situated in the left hypochondriac region, and extends vertically from the sixth to the ninth cartilage, and transversely from the fifth intercostal space in the parasternal line to the anterior axillary line. Its uppermost border forms a semicircle. It is bounded above by the diaphragm and the apex of the heart; upon the right, by the left lobe of the liver; and upon the left, by the spleen. This region is known as "**Traube's semilunar space.**" This space is of great importance in the differential diagnosis between consolidation of the base of the left lung and fluid in the left pleural cavity. Normally, this space has a well-marked tympanitic note upon percussion. If a moderate-sized pleural effusion occurs in the left pleural cavity, this space is encroached upon by the gravitation of fluid downward, and gives place to a dull or even a flat note. Consolidation of the base of the left lung does not give rise to this change.

Examination of the Liver.—The liver lies directly under the diaphragm, being held in place by the suspensory ligament, about three-fourths of it being in the right hypochondrium, and only about one-fourth extending over toward the left. The greater portion is covered by the peritoneum. During expiration it rises as high as the fourth interspace, falling again with inspiration. The gall-bladder lies within the right mammillary line, where the lower border of the liver passes under the right border of the ribs. In children the liver is proportionately larger in all its dimensions.

Inspection.—In healthy individuals no change in the contour of the abdominal wall is noted upon inspection; only in very young children in the first years of life there is a slight bulging in the right hypochondrium, due to the larger size of the liver owing to physiologic fatty infiltration. Only under circumstances in which the liver increases in bulk will it show signs upon inspection. In such instances the lower border of the liver may become displaced. This may also be due to displacement, and upon inspection it may be noted how the liver moves downward in deep inspiration. Occasionally, tumors may be observed in contact with the abdominal wall from the surface of the liver, and sometimes, although rarely, a distended gall-bladder may be seen.

Pulsation—due to an arterial, but more often to the venous, liver pulse—may be noted in the hypochondriac region. Increase in the size of the liver may occur from engorgement from fatty or amyloid infiltration, or from obstruction of the gall-bladder. It takes place in certain of the acute infectious diseases, such as relapsing fever. In malignant disease an irregular enlargement is found. The same is true of echinococcus, of syphilis of the liver, and of abscesses in this organ. Downward displacement occurs most often from causes that depress the diaphragm, such as severe chronic emphysema, pleural effusions, or pneumothorax affecting the right side. Subphrenic abscess may have a similar result. Occasionally, relaxation of the suspensory ligament may give rise to what is called the "wandering" liver.

Palpation of the Liver.—This is by far the most important method of examining the liver. The patient should be placed in the dorsal decubitus, and efforts made to have the abdominal wall relaxed. For this purpose the patient should be instructed to open the mouth and to breathe quietly. Deep breathing should be insisted upon in examining the lower

borders of the liver. Under normal conditions no part of the organ should be felt upon palpation. Tenderness upon palpation is a symptom of some importance. Many diseases of the liver occur without tenderness upon palpation, and it is only when the peritoneal layer becomes involved that pain and tenderness are apt to manifest themselves. Carcinoma usually causes pain, but it may exist without giving rise to tenderness. It is important upon palpation to feel for scars and tumors. Tumors appear in carcinoma, from echinococcus and from gummata. Scars are due most often to syphilis of the liver. Occasionally, in tubercular peritonitis small elevations may be felt at the lower border of the liver (miliary tubercles). Sometimes by a short, sharp stroke with the hand, a thrill is communicated to the other hand. This occurs from fluid in a cyst, and takes place in hydatid disease of the liver; it is known as the *hydatid thrill.* Under normal circumstances the gall-bladder can not be felt unless there is an accumulation of fluid, in which case it becomes accessible to palpation. Occasionally, when it is filled with gall-stones, they may be felt through the abdominal wall.

Percussion of the Liver.—The liver being an airless viscus, a flat note is obtained on percussion over the hepatic area wherever the liver comes in contact with the thoracic or abdominal wall. Anteriorly, liver dullness begins in the mammillary line at the sixth rib; in the axillary line, at the eighth rib; and in the scapular line, posteriorly at the tenth rib. It extends from all these points to the lowest border of the costal cartilages. Upward displacement of the liver may be due to a high position of the diaphragm. Enlargement of the liver occurs from many causes: from malignant disease, abscess, echinococcus, hypertrophic cirrhosis, etc.

Examination of the Spleen.—The spleen is situated in the left hypochondriac region, in the midaxillary line between the ninth and eleventh ribs. Under normal circumstances the spleen can not be palpated. Only in cases of enlargement is it possible to feel the spleen below the costal margin. Tenderness in the splenic region occurs in the infectious diseases, from infarcts, new formations, or from abscess. In some diseases the spleen attains enormous size. In malarial cachexia and in leukemia the spleen may occupy the entire left side and part of the right side of the abdomen.

TOPOGRAPHIC PERCUSSION.

The limitations of the special organs are represented by black dotted lines. The limits which appear dull upon deep percussion are represented by red lines.

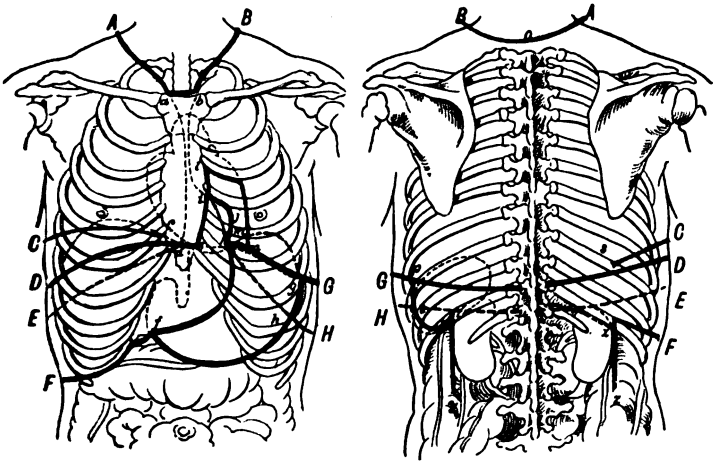

Fig. 9.

AaBb, Upper anterior area of normal pulmonary resonance.
DdeijkngG, lower anterior area of normal pulmonary resonance.
BoA, upper posterior area of normal pulmonary resonance.
GpqrD, lower posterior area of normal pulmonary resonance.
eijk, area of absolute cardiac dullness.
ilmnk, area of relative cardiac dullness.
Ffk, lower anterior border of absolute liver flatness.
Fx, lower posterior border of absolute liver flatness.
Cc, anterior border of relative liver dullness.
Cs, posterior border of relative liver dullness.
ptw, splenic dullness.
hmg, Traube's semilunar space.
wy, limit of dullness of left kidney.
xz, limit of dullness of right kidney. (*Modified from Wesener.*)

CLINICAL BACTERIOLOGY.

The following micro-organisms are commonly associated with suppuration and allied conditions: The staphylococcus pyogenes aureus, the staphylococcus pyogenes albus, the staphylococcus pyogenes citreus, the streptococcus pyogenes, the bacillus coli communis, the bacillus pyocyaneus, the diplococcus of pneumonia, the diplococcus intracellularis meningitidis, the ray fungus, the bacillus of glanders, and others. The gonococcus and the bacillus typhosus have also been found associated with suppurative conditions. Some lesions produced by the tubercle bacillus have a suppurative character.

Staphylococcus Pyogenes Aureus.—This micro-organism stains readily with the ordinary anilin dyes. It does not decolorize by Gram's method. This method of staining is as follows: (1) Stain the cover-glass preparation with analin-gentian-violet solution for half a minute; (2) wash in water; (3) stain with Gram's solution (iodin, 1 part; potassium iodid, 2 parts; water, 300 parts) for thirty seconds; (4) wash with alcohol until the stain ceases to come out of the specimen; (5) wash in water and mount. When examined microscopically, it is found in masses or clusters. Each organism is spheric in outline and measures about $\frac{9}{10}$ of a micron in diameter.

Biologic Characteristics.—Inoculation upon an agar slant reveals a growth in twenty-four hours, incubated at a temperature of 37° C. The colonies are smooth at first, have a shining surface, and grow along the inoculation stroke, the color at first whitish-yellow, later becoming orange. They are circular in outline, measuring about two millimeters in diameter. Uniform turbidity is produced when grown in bouillon. Stab cultures in gelatin reveal a growth in about twenty-four hours, liquefaction occurring in forty-eight or seventy-two hours, beginning at the top. Plate cultures reveal light-yellow, circular, and somewhat granular colonies, becoming darker as the growth advances. They are visible in about forty-eight hours after inoculation. Upon potato an abundant growth, having an orange color, is produced, growing at room-temperature. In litmus milk it is shown to be of an acid reaction, and coagulation is produced. The organism possesses marked resisting powers outside of the body. It requires a temperature of 80° C. for half an hour to produce death.

Staphylococcus Pyogenes Albus.—The biologic and morphologic characteristics of the organism closely resemble those

of the staphylococcus pyogenes aureus except that the growth appears white. Both are often present in the dust, in the air, and upon the surface of the body.

Staphylococcus Pyogenes Citreus.—The colonies of this micro-organism are of a lemon-yellow color. The virulence is less marked than the staphylococcus pyogenes aureus and albus; otherwise the characteristics are similar to the staphylococcus pyogenes aureus.

Staphylococcus Epidermidis Albus (Welch).—This organism is probably identical with the staphylococcus pyogenes albus. It is commonly found upon the surface of the body and is sometimes present in the deeper layers of the skin, being with difficulty reached by disinfecting agents applied to the surface. According to Welch, it liquefies gelatin more slowly, coagulates milk less rapidly, and is less virulent. It is a frequent cause of abscesses.

Streptococcus Pyogenes.—Upon microscopic examination it is found as a coccus somewhat larger than the staphylococcus aureus, about one micron in diameter; forming chains that vary in length, some being short, others, quite long. It does not decolorize by Gram's method, and stains readily with the ordinary anilin dyes.

Biologic Characteristics.—Its growth is slower than the staphylococci, and its vitality is not so pronounced. Upon agar it grows along the stroke in small, circular, semitransparent, white colonies, measuring about one millimeter in diameter. It grows at body-temperature. Gelatin stab culture reveals the growth along the puncture, which is white and appears about the second day. Liquefaction does not occur. It grows in bouillon. Its growth upon potato is not visible to the naked eye, but by microscopic examination of the material upon the surface it is found that the organisms grow. In litmus milk the growth possesses a faint acid reaction, and coagulation does not occur. Upon gelatin plates small, bluish-white, flat, rounded colonies appear in about seventy-two hours. Erysipelas is almost invariably due to the streptococcus.

Streptococcus Conglomeratus.—This micro-organism consists of masses made up by chains of cocci. Free chains are rarely seen. It differs from the streptococcus pyogenes in that cultures in bouillon grown at a temperature of 37° C. reveal a smooth, round, and very firm white scale, or a single layer in the bottom of the tube that is not disintegrated when

the tube is slightly agitated, while the streptococcus pyogenes forms a loose deposit in the bottom of the tube that is easily broken upon slight movement. "It has been found associated with scarlet fever" (Kurth).

Bacillus Pyocyaneus.—This organism measures about two microns in length and 0.5 micron in diameter. They are sometimes united in pairs or chains containing as many as six bacilli. The bacillus stains with the ordinary anilin dyes and not by Gram's method.

Biologic Characteristics.—It is motile. Upon agar slant it forms an abundant growth along the stroke, which is of a green color. When grown in gelatin, it rapidly produces liquefaction of the media, and assumes a green color throughout. When grown upon gelatin plates, colonies first appear as small white points, later becoming green. Liquefaction takes place around the colonies. The green pigmentation produced by this micro-organism only appears when grown in the presence of oxygen. According to Gessard's researches, two pigments are produced by this bacillus: one, a fluorescent green; the other, a blue pigment. Upon potato a yellowish-green growth appears.

Micrococcus Tetragenus.—This organism is a micrococcus measuring about one micron in diameter. It divides in two directions, so that groups of four (tetrads) are formed. It stains by Gram's method and readily with the ordinary anilin dyes. It grows upon culture-media, and does not liquefy gelatin. The colonies are of a yellowish-white color, circular in outline, granular, and slightly nodulated upon the surface. It is sometimes found in normal saliva, quite commonly in phthisical sputum, and in abscesses.

Diplococcus Intracellularis Meningitidis.—These micrococci occur in pairs, sometimes in fours, or in small groups, and when found in inflammatory exudates, they congregate, at times, inside of the pus-corpuscles. The organism is round or oval, resembling somewhat the gonococcus. It does not stain by Gram's method, but is demonstrated by Löffler's alkaline methylene-blue solution. At body-temperature it grows upon agar or glycerin-agar, forming colonies of a yellowish-brown color surrounded by a transparent zone. It does not grow well in bouillon or in blood-serum. It grows upon potato. This micrococcus was discovered by Weichselbaum in the exudates of cerebrospinal meningitis in 1887.

The Gonococcus.—This micrococcus is somewhat oval in

shape, and grows in pairs. The pair of cells may be compared to two beans placed side by side so that the concavities form the inner borders. This organism stains readily with the ordinary anilin dyes, and not by Gram's method. When found in pus, it frequently occupies the pus-cells, which is of importance in diagnosis. Artificial cultivation is difficult. It grows upon blood-serum, particularly human serum, at a temperature of from 30° C. to 34° C. The color of the growth upon blood-serum is found to be grayish-yellow, is very thin, scarcely visible, and reaches its maximum growth upon culture-media after two or three days, when no further development occurs, and it soon loses its vitality. The gonococcus is killed when exposed to a temperature of 60° C. This micro-organism was discovered by Neisser in 1879, obtained from gonorrheal pus. It is always present in the discharge from gonorrhea, whether it be from the genital tract or from other parts of the body. When affecting the mucous membrane, the gonococcus penetrates between the epithelial cells and is sometimes found in the layers beneath the epithelium. It is associated with urethritis, cystitis, suppuration of the kidneys, salpingitis, arthritis, endocarditis, and other conditions.

Diplococcus Lanceolatus (Fränkel's Pneumococcus).—These organisms exist as oval cocci, and are found in pairs, sometimes in chains. A transparent capsule is sometimes noted surrounding the pairs in stained preparation. It stains by Gram's method and with the ordinary anilin dyes. (Fig. 10.)

Biologic Characteristics.—It is easily cultivated on the ordinary culture-media, particularly when having a slight alkaline reaction, growing best at 37° C. or at room-temperature. It does not liquefy gelatin. The thermal death-point is 52° C. for an exposure of ten minutes. Upon blood-serum the colonies appear as transparent pellicles along the stroke of the needle. As a rule, it does not grow upon potato.

This micro-organism was described by Sternberg in 1880, he having inoculated some of his own saliva subcutaneously in rabbits, thus producing septicemia. Pasteur also described it in 1880. It is found associated with croupous pneumonia; this was demonstrated by Fränkel, Weichselbaum, Sternberg, and others. It is usually present in normal saliva, and is the most frequent organism found in the sputum of individuals suffering from croupous pneumonia. It has also been found in the inflammatory exudate in meningitis, ulcerative endo-

carditis, otitis media, and other conditions. In cases of croupous pneumonia caused by the diplococcus lanceolatus many of the general symptoms are due to the toxins eliminated from this organism; it is usually only confined to the lung.

The Bacillus of Friedländer.—This micro-organism is a short bacillus having rounded ends, often resembling a micrococcus; they are sometimes united in pairs or chains. A capsule surrounds the organism when preparations are made from blood inoculated with this micro-organism. This capsule can not be demonstrated when taken from cultures in artificial media. It stains quite readily with the anilin dyes, and decolorizes by Gram's method. It grows upon the ordi-

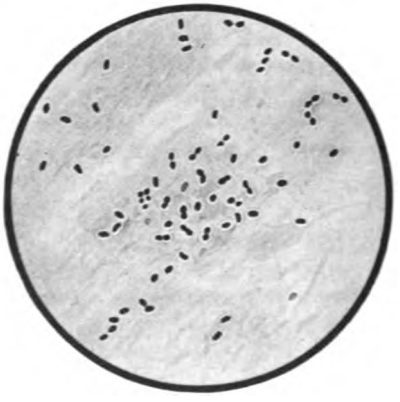

Fig. 10.—Diplococcus pneumoniæ; × 1000.

nary media. Upon gelatin plates at the end of twenty-four hours small white spheric colonies appear. The thermal death-point is 56° C. The organism is nonmotile and does not liquefy gelatin. It is aerobic, and facultative anaerobic. It grows upon potato. The colonies are yellowish-white in color. In gelatin stab cultures the growth presents a nail-like appearance, the line of the stab revealing a white growth, and the growth is heaped up upon the surface of the gelatin. Occasionally, gas is produced. This organism is sometimes associated with croupous pneumonia, but much less frequently than the diplococcus of pneumonia.

The Bacillus of Tuberculosis.—This micro-organism measures from one and one-half to three and one-half microns

in length, and about $\frac{2}{10}$ of a micron in diameter. The bacillus is straight or slightly curved (bent at an angle). It may be solitary, united in pairs, or sometimes in short chains; rarely, branching forms are met with. The staining reaction of the bacillus tuberculosis is quite characteristic inasmuch that it takes up stain with difficulty. Powerful staining solutions should be employed, as solutions of gentian-violet or fuchsin containing an anilin oil, or carbolic acid. The stain must be applied for a long period of time, or staining may be promoted by the application of heat. When the bacillus is stained, it resists decolorizing agents, such as 20% solutions of sulphuric or nitric acid. It is stained by Gram's method. A very satisfactory stain that is generally employed is Ziehl-

Fig. 11.—Bacillus tuberculosis in sputum; × 1000.

Neelsen's carbol-fuchsin (saturated alcoholic solution of fuchsin, 10 parts; and a 5% carbolic acid water, 90 parts). After staining with this solution the fluid is washed off with water, and then treated either with the strong solution (20%) of sulphuric or nitric acid until decolorized, or with Gabbet's methylene-blue solution for about thirty seconds. This solution consists of methylene-blue, 2 parts; sulphuric acid, 25 parts; and water, 75 parts. When using Gabbet's solution as a contrast stain, the tubercle bacilli will appear red, while other micro-organisms will be stained blue. The smegma bacillus and the lepra bacillus resemble the tubercle bacillus as regards staining reaction.[1] The latter can be differentiated from the smegma bacillus from the fact that it does not decolorize when

treated with alcohol. As a rule, this test need not be applied unless dealing with an examination of the urine. The tubercle bacillus is differentiated from the bacillus of leprosy in that it is somewhat shorter and retains the stain more firmly than the latter. Confusion between the two bacilli does not often occur.

Biologic Characteristics.—This bacillus shows marked resisting powers, and retains its vitality for a long time under various conditions. It this respect it resembles bacteria that form spores. The question of spore-formation, however, has not been determined in this organism, but it seems probable that it does form spores. After an exposure of two months sputum has been found to contain virulent bacilli. In the dried condition, when subjected to a temperature of 100° C. for an hour, the bacilli retain their vitality. The temperature of 70° C. in a moist chamber for a short time is usually fatal to the organism. A 5% solution of carbolic acid is fatal to the bacillus when exposed for five minutes. The organism can be cultivated upon blood-serum, a growth resulting in about two weeks that consists of small dried scales of a whitish or bluish color. The outline of the colonies is somewhat irregular and scattered over the surface of the media. They can also be cultivated in glycerin broth or glycerin agar, the most suitable temperature for growth being 37° C., but they can not be cultivated upon the ordinary gelatin or agar media.

This micro-organism was discovered by Koch in 1882. It is usually found in the sputum of those suffering from tuberculosis of the lungs, and often in the feces when tuberculosis of the intestinal tract is present; in the urine, when the infection occurs in relation to the genito-urinary apparatus. In the lesions produced by the tubercle bacillus it is commonly situated in the cheesy mass, giant cells, and epithelioid cells. When necrosis is well advanced, it is sometimes absent in the center of the caseous substance. Tuberculosis is one of the most common of all diseases met with, not only affecting man, but many of the lower animals, such as cattle, horses, birds, etc.

Koch, in 1890, introduced a substance called tuberculin as probably being of use as a curative agent. It has, however, proved itself to be of no value in this connection, but of great use in diagnosis. Tuberculin consists of filtered products from old fluid cultures, the bacilli having been killed by heat. Tuberculin when injected into healthy animals produces no ill effects,

but when injected into a tuberculous subject, produces a marked reaction, particularly the appearance of fever.

The Bacillus of Leprosy.—This bacillus measures from four to six microns in length and about one micron in diameter. It occurs in straight rods, sometimes bent or curved. It stains with the ordinary anilin dyes, taking up the stain with difficulty, but retaining it when treated with strong acid solution. It retains Gram's stain.

Biologic Characteristics.—Like the tubercle bacillus, this micro-organism stains with carbolfuchsin, and should be treated subsequently with a strong solution (20%) of nitric acid, or Gabbet's solution. (For points of differentiation between this organism and the tubercle bacillus refer to the Bacillus of Tuberculosis.)

This micro-organism has never been cultivated upon artificial media. The bacillus is nonmotile. Leprosy has been produced experimentally by the inoculation of the bacillus into criminals. It was first described by Hansen in 1871, and since that time the discovery has been confirmed by other observers.

The Bacillus Mallei (the Bacillus of Glanders).—This bacillus is straight or slightly curved, and about the same length as the bacillus of tuberculosis. It stains quite readily with the ordinary anilin dyes. It is easily decolorized by acid solutions, also by Gram's method.

Biologic Characteristics.—This bacillus is nonmotile, aerobic, and grows best at a temperature of 37° C. It can be cultivated upon various forms of artificial media. It loses its vitality when exposed to a temperature of 55° C. for five minutes, or when treated with a 5% solution of carbolic acid. Upon agar it produces an extensive growth in about two days. This growth is thick and white in appearance; after three or four days the color becomes amber yellow. Upon blood-serum it forms a yellow, transparent, drop-like colony. It also grows quite readily in bouillon, upon potato, and does not liquefy gelatin nor blood-serum. This bacillus was discovered by Löffler and Schultz in 1882, and was found in the tissues of animals suffering from glanders. The substance of use in diagnosis of glanders is known as mallein, which bears the same relation to glanders that tuberculin does to tuberculosis. Mallein is prepared from old glycerin bouillon cultures. This culture is filtrated, and the product is the substance used. When subcutaneously injected into animals

suffering from glanders, a reaction is produced in from four to ten hours.

The Bacillus of Anthrax.—The appearance of this bacillus, when obtained from bouillon cultures, is as follows: It grows in chains, each bacillus measuring from one to one and one-half microns in breadth, and from five to twenty microns in length. It is a straight bacillus with rounded ends, and it stains with the ordinary anilin dyes and also by Gram's method. It produces spores.

Biologic Characteristics.—It is a nonmotile aerobic micro-organism, and grows in a variety of culture-media at a temperature of from 20° C. to 38° C. Upon gelatin plates, colonies are developed that are irregular in outline and of a greenish color, developing in from twenty-four to thirty-six hours. Mycelium-like outgrowths are often seen extending from the periphery of the growth into the surrounding gelatin. When grown in gelatin, liquefaction takes place at the end of two or three days. Upon nutrient agar a grayish-white growth is produced at the end of twenty-four hours. When grown upon blood-serum, this media is liquefied. Spores are only produced in the presence of oxygen. At a temperature of 54° C., when exposed for ten minutes, this micro-organism loses its vitality if spores are not present; the spores, however, show great resisting powers. This bacillus was first obtained in the blood of infected animals by Pollender in 1849.

Bacillus Typhosus.—This bacillus measures from one to three microns in length, and from about $\frac{5}{1000}$ to $\frac{8}{1000}$ of a micron in breadth. Its ends are rounded. Around the periphery of the bacillus there are numerous flagella, numbering from five to twenty. These flagella are about five times the length of the bacillus, and about $\frac{1}{1000}$ of a micron in breadth. It stains with the ordinary anilin dyes and easily decolorizes. Special methods of staining are necessary to demonstrate the flagella, which are more numerous in the bacillus typhosus than in the bacillus coli communis.

Biologic Characteristics.—When this organism is inoculated upon agar media, colonies, circular in outline, are produced in about twenty-four hours, of a bluish-gray color when held to the light (transmitted light), and a dull white by reflected light. When grown in gelatin, liquefaction is not produced. It grows readily in bouillon. When examined in the hanging drop, it is found to be actively motile, old cultures being less

active. It develops best at a temperature of 37° C., and is killed after an exposure of half an hour at 60° C., and after two or three minutes at 100° C. Freezing does not destroy it. Upon potato, the growth is quite characteristic. At the end of twenty-four or forty-eight hours the surface appears velvety and of a dull white appearance. In litmus milk the culture is found to be alkaline in reaction, and coagulation is not produced. It does not produce gas when grown in agar media containing lactose, and does not produce indol. The bacillus was discovered by Eberth in 1880, who demonstrated its presence in the spleen and mesenteric glands.

The Widal Reaction.—When the blood of an individual suffering from enteric fever is mixed with a twenty-four-hour

Fig. 12.—Bacillus typhosus; × 1000.

old bouillon culture of the bacillus typhosus, a marked reaction occurs that consists in the loss of the motility of the micro-organism, and clumping or grouping of the bacilli. A drop or two of blood should be obtained. This is collected upon a clean sheet of paper or upon a glass slide, and allowed to dry. The dried film may be preserved for a long period of time. The dried blood should be dissolved with sterilized water and mixed with the bouillon culture in the proportion of one part of blood to from twenty to fifty parts of the culture and water. This mixture should be placed upon a cover-glass, and mounted upon the hollow-ground slide as a hanging drop. If the reaction be positive, stoppage of motility and clumping will occur during the course of half an hour; if

the reaction be negative, the bacilli will neither lose their motility nor clump. The reaction is found to be positive in about 95% of the cases of enteric fever. The blood in some diseases (miliary tuberculosis) occasionally gives the reaction. A pseudoreaction may occur, which consists in slight clumping and diminished motility. This is not indicative of enteric fever.

The Bacillus Coli Communis.—This micro-organism appears in short rods with rounded ends, the length varying somewhat between two and three microns, and its breadth measures about $\frac{5}{10}$ of a micron. It does not produce spores. The bacilli are sometimes linked in pairs or short chains. It is supplied with flagella, numbering from two to ten, and is motile, but not so active as the bacillus typhosus. It stains with the ordinary anilin dyes and decolorizes by Gram's method.

Biologic Characteristics. — This micro-organism grows readily upon the ordinary forms of culture-media, growing best at about 37° C., the thermal death-point being 60° C., when exposed for ten minutes. Upon agar and blood-serum, at the end of twenty-four hours a thick, moist, grayish-white layer is formed upon the surface. Upon gelatin plates colonies are noted at the end of twenty-four hours, and appear as spheric masses of a brownish color, the darkest shade being near the center of the colony. Superficial colonies are sometimes round or irregular in outline, and measure about three millimeters in diameter. The organism does not liquefy gelatin. Stab cultures in sugar agar and shake gelatin cultures produce gas-formation. When grown upon potato, a luxuriant growth, which is of a brownish-yellow color, is noticed at the end of twenty-four or forty-eight hours. When grown in litmus milk, coagulation appears, the media changing its color to red, demonstrating an acid reaction. The micro-organism also produces indol. It is normally found in the intestinal canal, and when it gains access to tissues, inflammatory lesions are produced. It is a frequent cause of peritonitis, appendicitis, and infections due to strangulation of the bowel, etc. It has been found in pneumonic exudates, pleurisy, and endocarditis. It is also associated with inflammations of the genito-urinary tract, such as cystitis. It is frequently met with in abscess-formation. A number of varieties of the colon bacillus have been described. It must be remembered that the bacillus typhosus closely resembles this organism, and by

some it is regarded that the two are indistinguishable; however, experiments have shown that the colon bacillus and the bacillus typhosus under similar conditions can not be transformed from one into the other, each organ retaining its own characteristics. The main differences between the bacillus typhosus and the bacillus coli communis are the following:

Bacillus Coli Communis.	*Bacillus Typhosus.*
Flagella fewer in number.	Flagella numerous and longer.
Slight motility.	Active motility.
Decolorizes by Gram's method.	Decolorizes by Gram's method.
Growth upon culture-media vigorous.	Growth generally less rapid and not so abundant.
Does not liquefy gelatin.	Does not liquefy gelatin.
Growth upon potato brownish-yellow and luxuriant.	Growth upon potato nearly transparent.
Acid production marked.	Produces an alkaline or very slightly acid reaction.
Coagulation marked.	No coagulation.
Indol production marked.	Indol production absent.
Gas production marked.	Gas production absent.
Clumping absent when mixed with typhoid serum.	Agglutination with typhoid serum positive.

The colon bacillus was obtained by Emmerich in 1885.

The Bacillus of Diphtheria.—This micro-organism appears as a bacillus having rounded ends, and measures about three microns in length and $\frac{5}{10}$ of a micron in diameter. It is straight or slightly curved, varying considerably, depending upon artificial cultivation. When grown upon agar, the organism is larger than when grown in broth. Upon serum and gelatin the organisms are of medium size. The bacillus stains with the ordinary anilin dyes, Löffler's alkaline methylene-blue solution readily staining the organism. The formula for this solution is as follows: Saturated alcoholic solution of methylene-blue, 30 c.c.; solution of caustic potash in water (1 : 10,000), 100 c.c. When stained with this solution, quite a characteristic appearance is noticed, the bacillus staining irregularly, there being areas that are deeply stained alternating with paler stained areas, giving the bacillus a beaded or dotted appearance; sometimes they stain uniformly. It is not decolorized by Gram's method. Spores have not been demonstrated.

Biologic Characteristics.—This organism is aerobic, but also facultative anaerobic. It is nonmotile, and grows upon the ordinary culture-media, but increases best, however, upon blood-serum, more rapidly upon this medium than other

micro-organisms. Upon blood-serum a luxuriant growth is noticed at the end of twenty-four hours, colonies developing that are round, elevated masses of a grayish-yellow color, the center being darker than the periphery, which is somewhat irregular. The bacillus does not liquefy gelatin, and grows slowly upon this medium. It produces an acid reaction during the earlier part of its growth, which later becomes alkaline. It does not produce gas. The indol reaction is present in cultures. Its thermal death-point is 54° C. when exposed for ten minutes. It grows best at 37° C.

This bacillus produces certain well-marked pathologic changes: the production of pseudomembrane, the most common situation of this being the pharynx, uvula, and tonsils;

Fig. 13.—Bacillus diphtheriæ; × 1000.

however, many other situations may be mentioned, such as the posterior nares, the bronchi, and the skin. It is found in the membrane, upon the free surface of it, and in the tissues beneath the membrane, but does not extend very deeply.

The toxins eliminated by this organism produce certain well-marked changes, the most common being cloudy swelling of various organs, particularly of the kidney, congestion of various organs, hyaline degeneration of the blood-vessels, and certain nerve-lesions.

The virulence of the diphtheria bacillus varies, and it should be remembered that this organism is often found in the mouth long after the inflammatory lesions have subsided.

This bacillus was first observed by Klebs in 1883, and

later, in 1884, by Löffler, who isolated the organism and demonstrated its pathogenic power.

Animals, especially horses, may be rendered immune by the gradual injection of diphtheria toxins.

The toxins are procured by filtering cultures possessing a high degree of toxicity through a Pasteur-Chamberlain filter. The first injection of the toxins into horses produces a marked reaction. The dose of toxin is gradually increased at each injection, and after some time the serum of the horse possesses marked antitoxic properties. This antitoxin is of great value in the treatment of diphtheria.

In the preparation of a curative serum, the toxin should be of such a strength that a guinea-pig weighing 250 grams will be killed on the fourth or fifth day after the injection. When ten times the minimum fatal dose of toxin is neutralized by the injection of 0.1 c.c. of antitoxin into a guinea-pig, this is designated as normal serum; 1 c.c. of this serum contains a *normal antitoxin-unit.* When 0.01 c.c. of antitoxin protects an animal from ten times the fatal dose of toxin, 1 c.c. of this serum contains 10 antitoxin-units. If only 0.001 c.c. is required, then 1 c.c. of such a serum represents 100 antitoxin-units.

Pseudodiphtheria Bacillus.—This bacillus possesses very slight pathogenic properties. It is sometimes found in anginal lesions. It measures between one micron and two microns in length, and the ends are rounded and slightly thicker than the remaining part of the bacillus. It stains with the ordinary anilin dyes, and the staining reaction is regular. It is stained by Gram's method. It does not form spores and is nonmotile. The growth upon agar, serum, and gelatin closely resembles that of the diphtheria bacillus. Slight growth is noticed upon ordinary potato. It does not produce gas in stab cultures. The growth produces an alkaline reaction. It is nonliquefying and does not coagulate milk. When grown in peptone media the indol reaction is produced in about three weeks. Sulphuric acid must be added to obtain the reaction. The indol reaction is obtained with the cultivation of the diphtheria bacillus in about a week. The organism is nonpathogenic to guinea-pigs when administered in doses of 5 c.c. of a forty-eight-hour broth culture.

The Bacillus of Tetanus.—This micro-organism appears as a straight, slender bacillus with rounded ends, measuring

about four or five microns in length, and $\frac{4}{10}$ of a micron in diameter, and when spores are formed it presents a thickening at one end, so that the organism may be compared to the shape of a pin or a drumstick. It is motile, possessing a number of flagella. It stains with the ordinary anilin dyes and by Gram's method.

Biologic Characteristics.—The organism readily forms spores. It is anaerobic, and grows best at the temperature of about 37° C. upon gelatin, agar, and blood-serum. The bacillus of tetanus possesses marked degrees of resisting power. The spores do not perish when exposed to a temperature of 80° C. for an hour in the dried state. When exposed to carbolic acid (1 : 20), it is destroyed only after fifteen

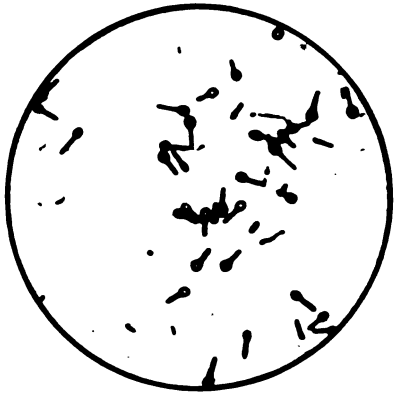

Fig. 14.—Bacillus tetani; × 1000.

hours. It must be cultivated in a stream of hydrogen, being distinctly anaerobic. The growth possesses a feeble alkaline reaction. The stab culture in gelatin reveals a growth in the central puncture that has an opaque appearance. It slowly liquefies the media, and may be grown at a temperature of 22° C. Sometimes a small amount of gas is produced around the puncture.

Kitasato in 1889 obtained the micro-organism in pure culture. Nicolaier in 1884 produced tetanus in mice by inoculation of garden earth, and also demonstrated that the disease could be transmitted by inoculation to other animals.

The toxins elaborated by the tetanus bacillus possess a high

degree of virulence. The quantity necessary to kill a mouse is 0.00001 c.c. of a filtrate obtained from bouillon culture.

The tetanus bacillus is found in garden soil, where it probably leads a saprophytic existence. It gains access to the body through a wound or a slight abrasion, the lower extremities being frequently affected. (Idiopathic tetanus probably does not exist, this term having been applied when the channel of infection had not been determined.)

Microscopic examination of scrapings or a section from a wound usually reveals numerous bacilli.

The tetanus bacillus is differentiated by its drumstick appearance when spore formation has taken place. There would be no doubt of the identity under such circumstances. If the wound be contaminated by other bacteria and the tetanus bacillus does not show spore formation, the diagnosis by the microscope will be impossible, and under such circumstances cultivation and inoculation experiments must be resorted to in order to demonstrate the bacillus.

The general symptoms of tetanus are due to the virulent toxins, which have an elective action upon the spinal cord. The toxic power of the urine is greatly increased in tetanus.

Antitetanic Serum.—This serum is made by the injection of toxins into a horse. The principles of preparation are about the same as those used in the making of the antidiphtheric serum. This serum is used in the treatment of tetanus; the results, however, have not been nearly so good as in the treatment of diphtheria by the diphtheria antitoxin.

The Bacillus of Malignant Edema.—This bacillus somewhat resembles the bacillus of anthrax in regard to size. It measures about 3½ microns in length and $1\frac{1}{10}$ microns in diameter. Sometimes the bacillus is much longer, measuring from 15 to 40 microns. The bacillus is straight, occasionally somewhat curved. It produces spores that are centrally located in the bacillus, producing a central swelling. It stains with the ordinary anilin dyes, and not by Gram's method.

Biologic Characteristics.—The bacillus is anaerobic, being best grown in a stream of hydrogen. It is motile. When grown upon nutrient agar at the ordinary room-temperature, the colonies are irregular in outline and present a white appearance. It liquefies gelatin and blood-serum. When grown on blood-serum, gas production is noted. This bacillus was first described by Pasteur, who called it "*vibrion septique*." It is associated with spreading inflammatory con-

ditions attended by edema and emphysema of the skin, often terminating in gangrene. This bacillus is found in garden soil and putrefying animal fluids. Immunity may be induced by the gradual injection of the toxins into susceptible animals.

Spirillum of Cholera.—This micro-organism appears in slightly curved rods resembling a comma, hence the name, "comma bacillus of Koch." Sometimes these are united in pairs, forming a circle or an S-shaped figure. The spirillum measures about one or two microns in length, and about $\frac{3}{10}$ or $\frac{4}{10}$ of a micron in diameter. It stains with the ordinary anilin dyes, and is decolorized by Gram's method.

Biologic Characteristics.—This spirillum is motile, possess-

Fig. 15.—Comma bacilli (from the mouth); × 1000.

ing a single terminal flagellum. It does not form spores, is aerobic, and grows upon the ordinary forms of culture-media, which should have a slight alkaline reaction at about room-temperature, but more readily at 37° C. The thermal death-point is 55° C. when exposed for one hour. A temperature of —100° C. does not destroy its vitality when exposed for several hours. On gelatin plates colonies appear in from twenty-four to forty-eight hours. They are granular, of a white color, and circular in outline. Liquefaction of the media soon occurs, giving the area a cup-like depression. In a gelatin stab culture liquefaction is produced, the micro-organism growing along the stroke. Upon blood-serum liquefaction of this media results. It grows upon potato at the temperature

of 37° C., producing colonies of a grayish-brown color. It does not coagulate milk. When grown in peptone media, and a few drops of sulphuric acid are added to the culture, a red color is produced, called the "cholera red reaction." This spirillum was described by Koch in 1884 in the excreta of a patient suffering from cholera, and was also found in the intestines of those having died of this disease. In patients suffering from cholera this micro-organism is confined to the intestines, occurring there in enormous numbers and in almost pure cultures. It has not been found in the blood or in the internal organs. Immunity may be induced in the guinea-pig by gradual injections of nonfatal doses of culture. The serum will give a specific agglutination test when mixed with the

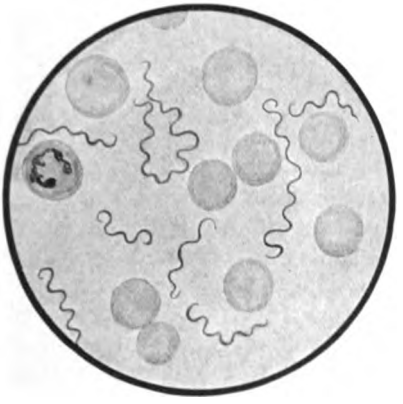

Fig. 16.—Spirilla of relapsing fever in human blood; × 1000.

pure culture of the cholera spirillum in the proportion of 1 : 20 to 1 : 100.

Spirillum of Relapsing Fever.—The spirillum of relapsing fever appears in the peripheral blood of patients suffering from this condition, during the febrile paroxysms, and disappears during the intermissions. It is a long, slender, spiral organism measuring from fifteen to forty microns in length, and possessing active motility. It is best studied by examining the fresh blood during the febrile paroxysm. It stains with the ordinary anilin dyes and decolorizes by Gram's method. This organism has never been cultivated upon artificial media. It has been kept alive for a few hours by placing the infected blood into tubes and sealed. The thermal death-

point is 60° C., but a temperature of 0° C. does not destroy their vitality. The disease has been produced experimentally by the inoculation of blood from individuals suffering from relapsing fever into apes, the period of incubation then being about three days, followed by a period of pyrexia that lasts from two to three days, ending by crisis. Usually no relapses result. Phagocytosis is clearly demonstrated in this disease. During the period of apyrexia the organisms probably accumulate in the spleen, where they are also destroyed. Obermeier in 1873 discovered the spirillum (spirochæte Obermeieri) in the blood of patients suffering from relapsing fever. The disease has been produced in the human subject by inoculation with the blood infected by this micro-organism.

Metschnikoff's Spirillum.—This micro-organism closely resembles the spirillum of cholera in its biologic and morphologic characteristics, except that when injected subcutaneously into pigeons, inflammatory swelling occurs with manifestations of septicemia, and death takes place within twenty-four hours, which does not occur with the spirillum of cholera. It does not give the agglutination test when mixed with the serum of patients suffering from cholera, showing that the anti-cholera serum exerts no properties against this organism. This micro-organism was obtained by Gameleia from the intestinal contents of fowls.

The Spirillum of Finkler and Prior.—This spirillum was obtained from the stools of certain cases of cholera nostras; however, its relationship to this disease has not been demonstrated. Morphologically, it resembles the spirillum of cholera. Its biologic properties are different, in that it grows more rapidly. Liquefaction is noted at the end of twenty-four hours. Individual colonies upon gelatin plates are often of large size and produce marked liquefaction. The cholera red reaction is not obtained from the growth in peptone solutions. A fetid odor is produced from the growth of this micro-organism.

The Bacillus of Influenza.—This micro-organism measures about $1\frac{1}{2}$ microns in length, and $\frac{3}{10}$ of a micron in diameter, appearing in straight rods with rounded ends. They are usually solitary, but may be united in groups of three or four. The bacillus stains with the ordinary anilin dyes, and is decolorized by Gram's method.

Biologic Characteristics.—This bacillus is aerobic. It does not form spores and is nonmotile. It grows at 37° C.,

colonies appearing upon blood-serum in twenty-four hours, which are circular in outline and almost transparent. It may be grown upon glycerin agar, drop-like colonies developing that are almost transparent, and do not tend to join together, but have a distinct property of remaining separated from one another. It is cultivated with difficulty in bouillon, growing more readily if blood be added to the media. It does not grow in gelatin. Its thermal death-point is 60° C. when exposed for five minutes. This bacillus was discovered by Pfeiffer in 1892 in patients suffering from influenza. It has been isolated from the bronchial and nasal secretion, from the lungs (in cases of pneumonia), and other organs, but it is

Fig. 17.—Bacillus pestis (Yersin).

rarely present in the blood. In cases of influenza the microorganism is most frequently present in the bronchial secretion.

The Bacillus Pestis.—This bacillus occurs in short rods with rounded ends, measuring $2\frac{3}{10}$ microns in length, and $1\frac{7}{10}$ microns in diameter; it is almost oval. It stains readily with the ordinary anilin dyes, but does not retain its color when treated by Gram's method. In stained preparations it is often noted that the ends are more deeply colored than the central portion, giving rise to the so-called "pole-staining." The bacilli are frequently found in chains.

Biologic Characteristics.—It is grown best at 37° C., but it may be cultivated at a temperature as low as 18° C. Its thermal death-point is 58° C. when exposed for several hours. The organism sometimes occurs encapsulated, but

this has not been noted by all observers. It does not possess motility. Upon agar media the colonies are circular in outline, slightly raised, and of a creamy color. They are somewhat granular, and the margins are quite regular. In stab gelatin cultures the medium is not liquefied, and the growth along the inoculation is not well marked. Gas is not produced when grown in sugar agar media. It does not coagulate milk, and produces a feeble indol reaction in broth cultures a week old on the addition of sulphuric acid. A well-marked acid reaction is noted when grown upon nutrient litmus agar. This bacillus was discovered independently by Kitasato and Yersin in 1894, during an epidemic at Hong Kong. The bacilli are found in the diseased lymphatic glands in great numbers, sometimes in the blood, and they have also been noted in the liver, kidneys, spleen, lungs (producing plague pneumonia), and in many other organs. Infection by this micro-organism produces a high mortality, not only in man, but also in certain animals, particularly rats and mice. Immunity in rabbits has been produced by the gradual injection of cultures that have been killed by heat; the serum of such immune animals possesses certain protective powers. Antiplague serum obtained from immunized horses has been employed with favorable results.

The Bacillus Icteroides.—This micro-organism measures between two and four microns in length and $\frac{5}{10}$ of a micron in breadth, and has rounded ends. It stains with the ordinary anilin dyes, but loses its color when treated by Gram's method.

Biologic Characteristics.—The bacillus is motile, having from four to eight flagella. It does not liquefy gelatin, and can be cultivated upon the usual forms of media, growing best at a temperature of 37° C. Upon an agar slant a growth will be noticed in twenty-four hours, the colonies being somewhat transparent and of a glittering gray color. On transferring the growth to a lower temperature for about twelve hours, the periphery of the colonies will exhibit a white or pearly-brown color, and are raised somewhat higher than the central portion; after a few days this growth becomes somewhat liquid, and may run down the sides of the media. Upon gelatin plates a growth will be noticed in twenty-four hours. Colonies appear as transparent points, and after the growth has existed for about seven days, the central portion of the colony becomes opaque. The cultures give a feeble indol reaction. When grown in milk, coagulation is slowly produced. Glu-

cose is fermented. The bacillus may also be cultivated upon blood-serum, potato, and in bouillon. This micro-organism was discovered by Sanarelli, who obtained it in cultures from cases of yellow fever in 1897. Sternberg in 1890 obtained a bacillus known as the bacillus "X," from various organs of cases suffering from yellow fever, which appeared to have some causative relationship to the disease. This micro-organism is probably identical with the one described by Sanarelli. The bacillus icteroides can be obtained from the liver and kidneys of persons suffering from yellow fever. Other micro-organisms may be associated with it. These are due to secondary infection. The mode of entrance of the specific organism of yellow fever into the body has not been determined. It has not been isolated from the bowel or from the black vomit. Intravenous inoculation of living or dead cultures into dogs causes infection in from a few hours to twenty-one days. Symptoms of diarrhea, anuria, loss of weight, and jaundice were noted. Nephritis, fatty degeneration of the liver, and hyperemia of the intestinal walls were found upon postmortem examination. Inoculations into man give rise to symptoms of yellow fever, as stated by Sanarelli. The agglutination test can be performed by mixing the serum of yellow-fever patients with the bacillus icteroides in the dilution of about 1 : 40. Immunized serum gives promise of favorable results as a prophylactic and curative agent.

Micrococcus Melitensis.—This micrococcus was obtained from the spleen in cases of Malta fever by Bruce. The organism is oval or rounded, and measures $\frac{5}{10}$ of a micron in diameter. It grows singly, in pairs, and sometimes in short chains. It stains with the ordinary anilin dyes and decolorizes by Gram's method.

Biologic Characteristics.—It is aerobic, nonmotile, and does not liquefy gelatin. It is said to possess flagella. Upon agar slants colonies will be visible about the third day. As the growth becomes older it changes to a pearly-white color. It may be cultivated in bouillon. Growth is not visible upon potato media. The agglutination test has been observed by mixing the serum of patients suffering from Malta fever with the micrococcus melitensis.

Actinomyces (Ray Fungus).—This micro-organism belongs to a higher group than the bacteria. It produces a disease known as actinomycosis, which is common in animals but also affects man. It has been found in various organs of the body.

The tissue lesions consist of granulation tumors and often the production of suppuration, the micro-organism being contained in the pus. It first appears as filaments of great length, and about $\frac{5}{10}$ of a micron in diameter. In colonies these filaments present an irregular network arranged in a somewhat radiating manner, and some of the filaments show branching. The actinomyces, when seen growing upon culture-media, reveals filaments growing upward, the protoplasm of which becomes segmented into spores. These spores, when they become free, may again give rise to the growth. At the periphery of the colonies there are also seen pear-shaped bodies that are formed by swelling of the sheath around the extremity of the filament. When stained by Gram's method, the filaments and the spheric bodies retain the stain, while the club-shaped bodies are not colored. The pear-shaped bodies are sometimes absent in the colonies. Outside the body the parasite is said to grow upon certain forms of grain, such as barley, the infection being conveyed in this manner. It may be cultivated upon agar at the temperature of 37° C., colonies appearing about the fourth day, which are circular in outline, of a reddish-yellow color, and somewhat raised; the media surrounding may be colored a brownish tint. Older cultures present a somewhat corrugated aspect. The organism may be cultivated under anaerobic conditions. It slowly liquefies gelatin, and may be cultivated upon potato.

LABORATORY METHODS.

EXAMINATION OF THE SPUTUM.

The sputum consists of those substances which are expectorated either by the efforts of coughing or hawking. It results from various diseases of the respiratory tract; practically all diseases of this tract are accompanied by coughing and expectoration. When coughing is attended by expectoration, it is called *productive;* and when unattended by expectoration, it is spoken of as *unproductive*. In children, in the aged, and in the insane, expectoration is sometimes not noticed, as in these cases it is often swallowed. The examination of the sputum should be carried on by means of macroscopic and of microscopic methods.

Macroscopic Examination.—Under this heading will be

considered the quantity, the form or consistence, the color, the reaction, and the specific gravity.

The Quantity of Sputum.—The quantity of sputum varies greatly, depending upon the pathologic lesions. It is scanty or even absent in the early stages of acute bronchitis, croupous pneumonia, miliary tuberculosis of the lungs, and in the first stages of asthma. It is more profuse in cases of acute bronchitis which have lasted for several days, also after croupous pneumonia has existed for some time; in well-marked caseous tuberculosis with cavity formation, abscess of the lung, and bronchiectasis. It may be profuse in caseous tuberculosis, chronic bronchitis (bronchorrhea), and in empyema that communicates with a bronchus. In many cases the sputum is apt to be more profuse in the early morning and at night; this is particularly true of chronic tuberculosis. Again, the quantity may be profuse at certain periods of the day, as in bronchiectasis or in cavity formation.

Form or Consistence.—*Serous Sputum.*—In this form the character of the expectoration is watery, and consists largely of serum. It occurs in edema of the lungs with or without inflammatory lesions. In this variety of sputum albumin is present in large amounts, and the material is often frothy.

Mucous Sputum.—This occurs as the result of catarrhal inflammation of the respiratory tract. It has a glassy, transparent appearance, the consistence varying somewhat, depending upon the amount of saliva mixed.

Mucopurulent Sputum.—This is the most common form of sputum, and consists of a mixture of mucus and pus. It is usually translucent, yellowish or greenish in color, and may appear in flakes or in flattened masses circular in outline, called nummular or coin-shaped sputum. It is common in the later stages of inflammation of the mucous membrane of the respiratory tract, in tuberculosis, particularly when cavity formation is present. In the latter condition it is apt to be nummular, and is sometimes spoken of as cavernous sputum. Mucopurulent sputum may occur in bronchiectasis. On allowing it to stand it often separates into three layers: an upper frothy layer; a middle purulent, slimy, and watery layer; and a lower layer consisting of pus or granular material.

Purulent Sputum.—This form of sputum, as the name indicates, is composed almost entirely of pus. It is present in empyema communicating with the bronchus, in abscess of the lung, and in some forms of bronchitis, also in abscess of the

liver when this communicates with the bronchus, or in subphrenic abscess existing under similar conditions. This sputum varies in consistence; most commonly, however, it is quite thick. It has a peculiar, sour smell, not unlike buttermilk. The odor may be very offensive, especially when decomposition sets in. When allowed to stand, it separates into two layers—the lower one being made up chiefly of pus-corpuscles, the upper one being fluid, due to pus plasma. Should the sputum be very frothy, a third layer may be noted upon the top, consisting of frothy material.

Bloody Sputum.—This sputum may consist entirely of pure blood, it may be blood-tinged, or the sputum may be intimately admixed with blood. It is usually large in amount, has a bright red color, shows its watery character, and may be frothy. It most frequently occurs in tuberculosis of the lungs, appearing early or late with cavity formation. It is also met with in abscess and gangrene of the lung, infarction, trauma, croupous pneumonia, rarely in bronchiectasis, rarely in putrid bronchitis (from excessive coughing), tumors and aneurysms communicating with the bronchus and echinococcus, also in blood dyscrasias, such as purpura hæmorrhagica, and hemophilia, and it may occur in vicarious menstruation. Blood-tinged sputum may appear as the result of any of the conditions just enumerated, and sometimes follows copious expectoration of blood, this being due to a certain amount of blood that is retained in the air-passages. Blood-tinged sputum may also result from croupous pneumonia. It is of two characters—the first being known as rusty sputum, being very tenacious; and the second, "prune-juice" sputum, which is viscid and fluid, the color corresponding to prune juice, hence the name. It may appear in hemorrhagic infarcts, mitral disease, and in miliary tuberculosis. It is occasionally found in acute bronchitis, particularly in plethoric subjects.

Color of the Sputum.—This may vary, depending upon the cause. It may be translucent and almost colorless, or light yellow, such as occurs in chronic bronchitis. It may be of a deep yellow color when it is bile-stained, occurring in jaundice; or it may be red when containing blood or iron; or blackish gray, as a result of anthracosis. In some instances it is green, as a result of admixture of pus, particularly when the bacillus pyocyaneus is present.

Reaction.—The reaction of sputum is always alkaline.

Specific Gravity.—The specific gravity is from 1004 to

1026. Neither of the last two characteristics mentioned are of special diagnostic import.

Microscopic Examination of the Sputum.—Fibrinous casts of the bronchi are sometimes present in the sputum, and occur as a result of fibrinous or plastic bronchitis. They appear in the sputum as small spheric masses, and when placed in water, separate after being teased out into twig-like bodies. They are made up of fibrin, epithelial cells, some leukocytes, and even micro-organisms.

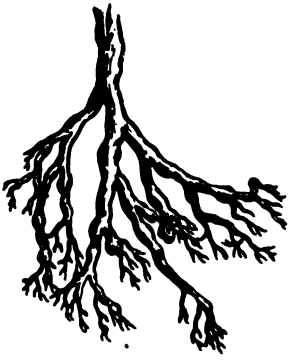

Fig. 18.—Fibrinous bronchial cast.

Spiral bodies are sometimes observed in the sputum. They were described by Curschmann and Leyden, and are known as Curschmann's spirals. They occur in asthma, bronchial catarrh, and fibrinous pneumonia. They vary in length and diameter, often being visible to the naked eye. They are frequently covered by epithelial cells and Charcot-Leyden crystals. They are composed of a substance resembling mucin.

Fig. 19.—Sputum from a case of asthma, showing Curschmann spirals, Charcot-Leyden crystals, leukocytes, and numerous free eosinophile granules; unstained specimen (Jakob.)

Dittrich's plug occurs in putrid bronchitis and gangrene of the lungs. These vary in size from that of a pinhead to that of a bean, being composed of fatty acids, fat corpuscles, round cells,

red blood-cells, hematoidin, and micro-organisms. Small, gelatinous, round masses are met with in the sputum of bronchial asthma, and are known as the *perles of Laennec*. Elastic fibers occur, particularly in tuberculosis, abscess, and gangrene of the lung. They vary in length and breadth, are usually curved, and are somewhat flattened. They are indicative of the destruction of lung-tissue. They frequently show an alveolar arrangement, and epithelial cells may be found adhering to these structures. They are only of diagnostic import when they conform to an alveolar arrangement. They may be detected by the following method: The sputum is mixed with a solution of caustic potash (8% to 10%), this is brought to the boiling-point, and then allowed to stand for twenty-four hours in a conical glass, when the sediment can be examined microscopically. Various other particles may be present in the sputum, such as fragments of cartilage, connective-tissue masses, or small portions of tumors (sarcoma and carcinoma).

Crystals.—*Charcot-Leyden Crystals.*—These vary in size, are elongated octahedrals, and of a transparent bluish color. They are easily visible with the low powers of the microscope. They are met with in asthma, sometimes in tuberculosis, croupous and acute bronchitis.

Cholesterin Crystals.—These appear as large, sometimes irregular, rhombic plates, showing a tendency to group. When treated with sulphuric acid, they become yellow and finally green; or with diluted sulphuric acid and tincture of iodin, a violet color is produced, which later changes to blue, green, and red. They are of little significance and have been met with in tuberculosis and abscess of the lung.

Hematoidin Crystals.—These have been found in the sputum as rhombic prisms or as needles, frequently in clusters. When these crystals are present, they are often indicative of recent hemorrhage. Fat crystals are encountered in putrid bronchitis and gangrene.

Triple Phosphate Crystals.—Triple phosphates are sometimes encountered in the sputum.

White Blood-corpuscles.—These are always present in the sputum, and large amounts are indicative of inflammatory conditions. The polynuclear eosinophiles are present in large numbers in asthma.

Red Blood-corpuscles.—These are also usually present in small numbers. When occurring in considerable quantity, they are indicative of hemorrhage or acute inflammatory con-

ditions. The blood-cells may be only slightly altered (crenated), or washed out (phantom or shadow corpuscles).

Epithelial Cells.—Sputum always contains some epithelium. When occurring in large numbers, this is indicative of pathologic changes, such as catarrhal inflammation, tuberculosis, etc. It may be squamous, columnar, or ciliated. Large, flat epithelial cells may arise from the alveoli. It must be remembered that under pathologic conditions epithelium may undergo alteration, especially as to shape; particularly is this so in bronchiectasis. The epithelial cells frequently reveal degenerative changes, so that the nucleus is with difficulty differentiated.

Micro-organisms.—Many micro-organisms are encountered in the sputum, and it may be said that the sputum always contains them when it is admixed with saliva.

The Bacillus of Tuberculosis.—This micro-organism is of great diagnostic import when found in the sputum, and is always indicative of tuberculosis. The material for examination is placed upon a glass plate, and then selecting the small cheesy masses contained therein, are placed upon a slide or cover-glass, spread out and gently dried over a flame; the material is then fixed by rapidly drawing the cover-glass through the flame three times. (For method of staining the tubercle bacillus, see p. 105.)

The Diplococcus of Pneumonia.—This micro-organism is found in the sputum in the greater number of cases of croupous pneumonia, and often in normal saliva. (For method of detection, see p. 103.)

The Bacillus of Influenza.—The bacillus of influenza is found in cases suffering from influenza. (For method of detection, see p. 118.)

The Bacillus of Diphtheria.—The bacillus of diphtheria may be present in the sputum of cases suffering from diphtheria. (For method of detection, see p. 111.)

The Bacillus of Friedländer.—The bacillus of Friedländer may be found in the sputum of some cases of croupous pneumonia. (For method of detection, see p. 104.)

The Ray Fungus.—The ray fungus is sometimes present in the sputum. (For method of detection, see p. 121.)

Animal parasites are sometimes present in the sputum, such as the echinococcus, the distoma pulmonale, and infusoria.

EXAMINATION OF THE STOMACH-CONTENTS.

The most important chemic constituents found in the gastric contents are pepsin, rennet or milk-curdling ferments, inorganic and organic acids. Before making a careful examination of the stomach-contents it is necessary to give a test-meal. This should be given upon an empty stomach; it is best, therefore, to administer the meal in the morning or after washing out this organ. Ewald's test-meal, the one most generally employed, consists of two cups of tea, without sugar and milk, or 300 c.c. of water, and 70 grams of dry wheat bread. The bread should be thoroughly masticated. The stomach-contents are withdrawn one hour after ingestion, at which time the maximum secretion of hydrochloric acid is obtained under normal conditions. This meal sometimes exerts very little stimulation to the stomach, particularly the secretion of gastric juice, and for this reason a test-meal of meat and starches has been suggested. Leube's test-meal consists of a plate of water soup, a small amount of beefsteak, and wheat bread. This test-meal is withdrawn from four to five hours after ingestion.

Method of Evacuating the Stomach.—Soft stomach-tubes are used for this purpose, the best being the Ewald, Leube, and Jaques. A mark appears upon the tube indicating the distance it should be introduced, which is from eighteen to twenty-two inches from the incisors. Before introducing the tube it should always be moistened with water, glycerin, or oil.

Method of Introducing the Tube.—The patient preferably should assume the sitting posture, and raise the head a little. The operator standing behind or to the right or left of the patient, grasps the tube in the right hand. The patient opening the mouth wide the tube is introduced until it reaches the end of the tongue; it is then rapidly but gently pushed downward, until the proper depth has been reached, the operator at the same time asking the patient to swallow. The first operation is usually attended with vomiting, excitement, disturbed breathing, and some cyanosis. These may be so great that the operation must be suspended. If the manœuver be repeated a number of times, the patient is usually able to introduce the tube himself. After the tube is inserted the contents may spurt out of it, or in other instances it is necessary to make compression in the region of the abdomen corresponding to the stomach, or have the patient bend forward. The stomach-pump may be employed, or the material may be

siphoned by allowing a quantity of water to flow into the tube, and lowered while it is well filled with fluid. The contraindications to the use of the stomach-tube are severe hemorrhages from the stomach and aneurysms. If great debility exists, it is sometimes better to strenghten the patient before performing the operation.

Gross Appearance of the Stomach-contents.—The contents should be carefully examined, the various solid particles noted, such as articles of food, pieces of tissue from the stomach-wall, bile, pus, blood, fragments of tumors, etc. Microscopic examination will determine the presence of pus, blood, or epithelial cells; the latter, if occurring in masses, presenting an irregular or atypical arrangement, strongly indicate carcinoma. Other solid particles that have been ingested may also be determined by microscopic examination; also micro-organisms, such as sarcinæ, fungi, and various bacteria. The Oppler-Boas bacillus is often found in cases of carcinoma of the stomach, particularly when lactic acid is present. The capacity of the normal stomach is about 1700 c.c.

Chemic Examination of the Stomach-contents.—After the Ewald test-meal has remained in the stomach for an hour it is withdrawn by the soft rubber stomach-tube in the manner previously described. About thirty cubic centimeters of the semidigested material is filtered and subjected to chemic examination; the following tests are of the most importance: The reaction, the total acidity, the presence of free hydrochloric acid, of lactic acid, of butyric acid, and of acetic acid. It is sometimes important to estimate the quantity of hydrochloric and lactic acids. The test for pepsin and rennet is also sometimes important.

Reaction.—Under normal circumstances the reaction of the material for examination is determined with litmus paper. Free acids, both organic and inorganic, may be tested with Congo-red paper, a blue color being noted if they are present.

Total Acidity.—This test is performed in the following way: Ten cubic centimeters of the filtered fluid are treated with a decinormal solution of caustic soda (4 grams of caustic soda to 1000 grams of water), using phenolphthalein as the indicator. Under normal conditions it requires from 50 to 65 c.c. of a decinormal solution of soda to neutralize 100 c.c. of filtered stomach-contents, the material being withdrawn one hour after the ingestion of Ewald's test-meal.

Tests for Free Hydrochloric Acid.—*Günzburg's Test.*—The reagent necessary to perform this test is the following: Phloroglucin, 2 grams; vanillin, 1 gram; and alcohol, 30 c.c. A few drops of the filtered stomach-contents are placed in a porcelain dish, and are mixed with the same amount of the reagent; if free hydrochloric acid be present, a rose-red color is developed. This test is quite delicate. Von Jaksch detected 0.001 n of acid in 10 c.c. of gastric juices.

Boas' Test.—The reagent necessary for this test is tl lowing: Resorcin, 5 grams; cane-sugar, 3 grams; and alcohol, 100 c.c. A few drops of the filtered stomach-co are placed in a porcelain dish, mixed with a few drops reagent, and evaporated to dryness. If free hydrochlor be present, a red color is developed; upon cooling the disappears.

Benzopurpurin Test.—Test-papers are prepared by s filter-paper in a watery solution of benzopurpurin, an allowing them to dry. On the addition of free hydro acid the color changes to a dark red. Acetic, formic, an acids give a similar reaction; the color, however, brownish violet.

Tropeolin Test.—Test-papers may be prepared by s filter-paper in a solution of tropeolin, and on the addition of hydrochloric acid a dark-brown color is produced. This test also responds to free lactic acid.

Lactic Acid.—*Uffelmann's Test.*—To a mixture of 20 c.c. of water and 10 c.c. of 4% watery solution of carbolic acid is added one drop of the tincture of the perchlorid of iron; an amethyst-blue color is developed. On the addition to this mixture of filtered stomach-contents that contain lactic acid, a canary-yellow color is noted. The delicacy of this test is hindered by phosphates, alcohol, glucose, and hydrochloric acid.

Boas' Test.—To about 20 c.c. of filtered stomach-contents add carbonate of barium,—in excess if the reaction of the fluid for examination be acid, and evaporate the mixture to a syrupy consistency; then add a few drops of phosphoric acid, heat the fluid so as to drive off the carbonic acid, allow the substance to cool, and pour 100 c.c. of ether (alcohol free) into it; shake the mixture, then allow it to stand for half an hour, when the ether is poured off and evaporated to dryness, the residue being dissolved in 45 c.c. of water and filtered. The fluid is poured into a Florence flask, a little manganese and 5 c.c. of

sulphuric acid added. A glass tube is used to connect the flask with a cylinder containing about 10 c.c. of an alkaline iodin solution (equal parts of a decinormal iodin solution and a decinormal solution of sodium). Upon heating the flask iodoform is formed in the iodin solution, which is detected by

ERRATUM

Page 130, fifth line of text, the sentence should read: "And are mixed with the same amount of the reagent and slowly evaporated to dryness."

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Free Hydrochloric Acid.—*Mintz's*
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nixture from time to time by placing
a few drops upon a porcelain plate and subjecting this to the phloroglucin vanillin test. The alkali is added to the filtrate until the hydrochloric acid is neutralized, this being determined when the mixture ceases to give the reaction with Günzburg's test. Note the number of cubic centimeters of decinormal solution of soda used and multiply this number by 0.00365 gram, the result being the amount of hydrochloric acid (expressed in grams) present in 10 c.c. of stomach-contents.

Quantitative Test for Lactic Acid.—After boiling 10 c.c. of filtered stomach-contents a few drops of sulphuric acid are added in order to coagulate the albumin; the solution is then filtered and evaporated to a syrupy consistency; to this are added 10 c.c. of distilled water, and the mixture evaporated to dryness; to the residue ether is added, the ethereal solution separated and evaporated; the remaining solid portion is dissolved in water and neutralized with a decinormal soda solution One cubic centimeter of soda solution is found to correspond to 0.009 gram of lactic acid. To complete the test, the number of cubic centimeters of soda solution used is multiplied by 0.009, the result being the amount of lactic acid present in 10 c.c. of the stomach-contents.

Test for Pepsin.—This test depends upon the property of pepsin changing fibrin into peptone. To 20 c.c. of filtered stomach-contents is added a small amount of hydrochloric acid, if the fluid is not acid in reaction. A small quantity of washed-out blood fibrin is placed into the fluid for examination and set aside in a warm place (40° C.) for a number of hours, and if pepsin be present, the fibrin will become dissolved; if after ten or twelve hours this does not occur, it may be inferred that the substance is not present. From the rapidity of solution the approximate amount of pepsin may be determined.

Rennet.—To indicate the presence of this ferment 10 c.c. of the filtrate are neutralized. To this is added an equal volume of sterile milk; after an exposure for twenty or thirty minutes at a temperature of from 30° to 40° C., coagulation will ensue if rennet be present.

Rate of Absorption of the Stomach.—This may be determined by administering a capsule containing 0.1 gram of potassium iodid, and after a short time testing the saliva with starch paper every two or three minutes; when iodin is present in the saliva, the moistened starch paper will assume a blue color on the addition of nitric acid. Under normal conditions this reaction appears in from eight to fifteen minutes after the capsule is swallowed.

The activity or motor power of the stomach is determined by administering a capsule of one gram of salol, this being decomposed into salicylic acid and phenol in the small intestine, where absorption takes place, salicyluric acid soon after appearing in the urine. This is demonstrated by the addition of a solution of perchlorid of iron to the urine, a violet color developing. Under normal conditions it requires from forty to sixty minutes for the stomach to expel its contents into the small intestine.

The Vomit.—The quantity and frequency of the vomit depend upon the amount of food ingested, and the pathologic lesions, such as gastritis, gastro-enteritis, peritonitis, tumors, some of the infectious diseases, uremia, diseases of the brain, and pregnancy. The character of the material vomited depends upon the food ingested and the degree of digestion, whether it be mixed with bile, pus, blood, or mucus.

Mucous vomit is encountered in chronic gastritis, nervous dyspepsia, and the dyspepsia resulting from a healed gastric ulcer.

A **serous vomit** occurs in Asiatic cholera (rice-water vomit).

Bile-stained vomit is due to the mixture of bile with the stomach-contents, and occurs from obstruction of the bowels, peritonitis, and sometimes from violent vomiting spells.

Blood Vomiting (Hematemesis).—This is due to congestion of the stomach, such as is encountered from portal obstruction, from hyperemia, and from inflammation; occasionally, blood is swallowed and then vomited. It is met with in yellow fever, in the hemorrhagic diathesis, in melena neonatorum, in rupture of an aneurysm, in ulceration of the stomach-wall, as in gastric ulcer and carcinoma, in vicarious menstruation, and in trauma.

Pus.—The vomiting of pus is observed when an abscess perforates into the stomach, or in exceedingly rare instances from phlegmonous gastritis.

Fecal Vomiting.—This is encountered in general peritonitis, and from obstruction of the bowels.

Animal parasites are sometimes encountered in the vomit, such as the oxyuris vermicularis, the anchylostoma duodenale, the trichina and echinococcous hooklets.

EXAMINATION OF THE BLOOD.

The blood is often of great importance in diagnosis; as it is a fluid tissue bearing a relation to other tissues, it reflects morbid conditions of distant parts. During the last few years methods of examining the blood have been improved, and this has lent additional accuracy to diagnosis in many important diseases. For all practical clinical purposes the examination of the blood consists of a macroscopic, a microscopic, and in some rare instances of a special chemic examination.

Method of Procuring the Blood.—Certain precautions are necessary in procuring the blood. For the purposes of ordinary examination the superficial blood is selected. It is rarely justifiable to secure the blood from an internal organ, such as the spleen. The lobe of the ear or the finger-tips are usually the points chosen. The former site is preferable since the skin is not so sensitive, is thinner, and the part more flexible; the manœuver can often be performed without the patient observing the operation. In hysteric women the lobe of the

ear should always be selected. Before making the puncture it is safer to inquire whether the patient is a bleeder, for if this is the case, the wound should be made with great precaution, and be very superficial, the operator having measures at hand to control the flow of blood. Great care must be taken not to select a part of the body that is edematous, for under such a condition the serum in the lymph-spaces gives rise to fallacies, such as dilution. It is also important to select a part where the circulation is good, the part being warmed by friction; inflamed areas should be avoided. Ordinary cleansers—soap and water—are all that are necessary in the preparation of the part, and attempts at careful sterilization are unnecessary. If the hand is selected for the site of puncture, it is advisable under ordinary circumstances to select the left hand if the patient is right-handed, or vice versâ. While septic infection has never been recorded from puncture in this manner, it requires no extra labor to use this precaution. If the skin is cold, rubbing so as to excite hyperemia is recommended by most authorities. A deep thrust, going into the skin ⅛ of an inch or slightly more is usually sufficient. The needle should have a cutting edge, such as the two- or three-sided surgical needle, or a steel pen or sharp instrument may be employed for this purpose. The first few drops should not be utilized for examination, but these may simply be wiped away. The part must not be squeezed, as by this procedure the serum will escape before the corpuscles.

Macroscopic Appearance of the Blood.—Color.—The color of the blood should be carefully noted. Blood, procured by puncture as just indicated, in the normal individual is bright red. If taken directly from an artery, it is of a brighter red color than when taken from a vein, the latter being bluish-red, the arterial blood containing more oxygen. In severe anemias the shade of the blood is found to be much lighter, and presents a serous character. In pernicious anemia it is frequently brownish-red in color, resembling thin coffee. It is dark red or bluish-red from cyanosis and during a chill. From poisoning by potassium chlorate, nitrobenzol, or amyl nitrite it is brownish-red. From coal-gas poisoning it is a scarlet-red color. In leukemia the blood often has a milky or pus-like appearance, and in lipemia it may have a similar appearance.

For determining the color of the blood it should be placed

upon a clean sheet of white paper and compared with normal blood.

Coagulability and Fluidity.—According to Vierordt, normal blood will coagulate in 9.28 minutes. The coagulability is increased after the administration of calcium chlorid and carbonic acid; also in some diseases, such as tuberculosis of the lungs, scurvy, and leukemia. It is decreased after the administration of citric acid and alcohol, and after increased respiratory efforts. In the acute exanthematous diseases and in the hemorrhagic diathesis it is also decreased; this is particularly true of hemophilia, it often requiring from thirty to fifty minutes for coagulation to take place in this condition.

Specific Gravity.—The specific gravity of normal blood is about 1059. The degree of specific gravity and the amount of hemoglobin seem to bear a distinct relation to each other, so that a decrease in specific gravity indicates a decrease in hemoglobin.

Hammerschlag's investigations [1] have proven that there is a much closer relationship between the specific gravity and the hemoglobin than between the specific gravity and the number of erythrocytes. The following table, based on many repeated examinations, has been found almost constant:

Specific Gravity.	*Hemoglobin (after Fleischl).*
1033 to 1035	25 to 30%
1035 " 1038	30 " 35%
1038 " 1040	35 " 40%
1040 " 1045	40 " 45%
1045 " 1048	45 " 55%
1048 " 1050	55 " 65%
1050 " 1053	65 " 70%
1053 " 1055	70 " 75%
1055 " 1057	75 " 85%
1057 " 1060	85 " 95%

Method of Determining the Specific Gravity.—*Hammerschlag's modification of Roy's method* is best suited for clinical purposes. It is based on the principle that a drop of blood is suspended in a liquid having the same specific gravity. Chloroform is heavier than blood and benzol is lighter, and when a mixture of these two liquids is obtained corresponding to the specific gravity of normal blood the latter will be suspended in the mixture. If the blood is lighter than the mixture, it will float; some benzol should be added until it is suspended. If it is heavier, some chloroform should be added. The

[1] "Centralbl. f. klin. Medicin," 1891, No. 44.

specific gravity of the mixture is then determined by the ordinary urinometer. Care should be taken to prevent air from gaining access to the drop of blood.

Alkalinity.—Normal blood is always alkaline in reaction. No practical method for determining the degree of alkalinity of the blood has yet been devised.

Estimation of Hemoglobin.—Under normal conditions the hemoglobin is contained in the red blood-cells as an albumin-

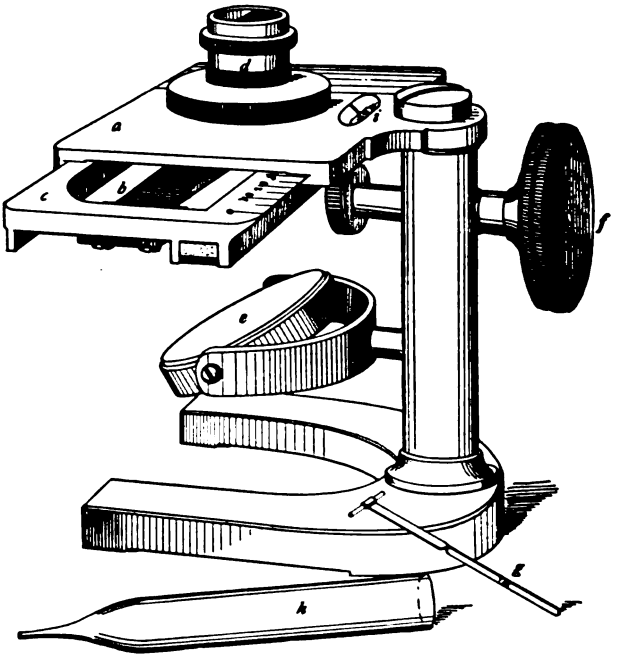


Fig. 20.—Von Fleischl's hemoglobinometer: *a*, Stand; *b*, narrow wedge-shaped piece of colored glass fitted into a frame (*c*), which passes under the chamber; *d*, hollow metal cylinder, divided into two compartments, which holds the blood and water; *e*, white plate from which the light is reflected through the chamber; *f*, screw by which the frame containing the colored glass is moved; *g*, capillary tube to collect the blood; *h*, pipet for adding the water; *i*, opening through which may be seen the scale indicating percentage of hemoglobin.

ate, which is soluble in water. For methods of determining the amount color tests are employed by comparing the blood with a color scale.

Von Fleischl's Hemoglobinometer.—The principle of this apparatus is based upon the fact that a mixture of l od and

water is compared with a graduated color scale, this being a wedge-shaped tinted piece of glass. The intensity of the color scale corresponds to certain percentages. A pipet for measuring the quantity of blood is used that is supplied with the apparatus. When this is filled with normal blood and mixed with a certain quantity of water, as will be described below, the intensity of the color should correspond to the color in the tinted wedge marked 100%. The cylinder in which the blood is mixed is divided into two compartments—one containing the mixture of blood; the other, distilled or pure water. This receptacle is then placed upon the stage of the instrument, the wedge-shaped tinted glass being directly under the half of the cylinder that is filled with water. A plaster-of-Paris reflector is used to reflect the light. The estimation must always be performed in a dark place, yellow light only being used for illumination, as from a candle.

A box may be constructed so that all but the adjustment of the instrument is in a dark space, artificial light being used for illumination. In matching the colors it is best to first move the thumb-screw to the light or the dark shade of the red, and then match the colors, the percentage being noted. This manœuver is repeated, starting from the opposite side of the color scale, the percentage again being noted. The average of the two readings should be the result. There is always a certain liability to error, so that the result is not absolutely accurate. Some individuals have difficulty in matching the colors. It is to be remembered that we must match the central portion of the color scale, one of its ends in view being lighter and the other darker. By using a shield that allows only a small portion of the central part to be in view error is almost avoided. When the percentage of hemoglobin is under 20, the reading is difficult and unreliable, so that in this case it is best to use two pipets filled with blood and divide the result by 2.

Oliver's Hemoglobinometer.—This instrument is also based upon a color comparison, except that it differs from the Fleischl instrument inasmuch that reflected light is used instead of transmitted light, also that in its percentage determination twelve tinted discs are used instead of a wedge-shaped tinted glass. These tinted discs are graduated so that they correspond to the number scale 10%, 20%, 30%, 40%, 50%, 60%, 70%, 80%, 90%, 100%, 110%, 120%. The blood is measured in a pipet similar to that used in the Fleischl apparatus, except

that it is much stronger and more easily handled and cleansed. The blood is then washed into the mixing cell and covered with a glass plate. Two forms of instruments are made, one to be used with candle-light, the other with daylight, the former being the more accurate. In order to estimate the percentage of hemoglobin more accurately—that is, so as to get the percentage nearer than 10 points—colored glass plates called "riders" are used, being placed upon the primary discs, which intensify the shade of the color. The error in this instrument is about 2%.

Gower's Hemoglobinometer.—This instrument is based upon a color comparison, the blood being diluted with water

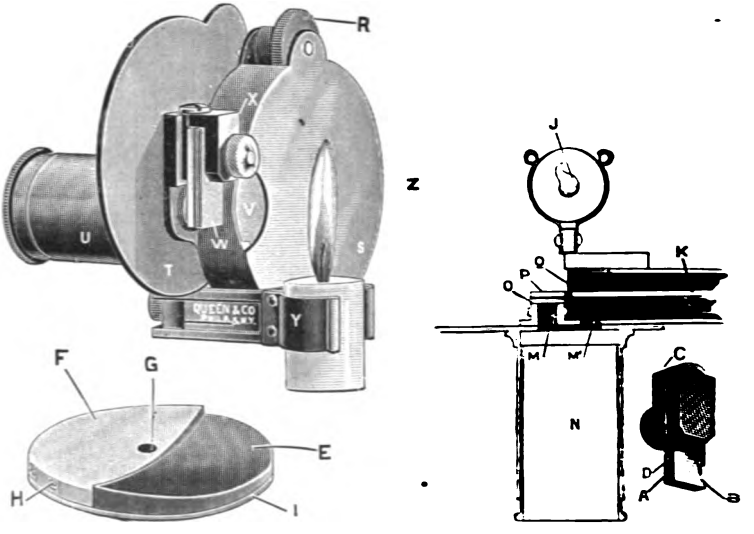


Fig. 21.—Dare's hemoglobinometer.

until it corresponds to a color standard. The amount of fluid required to dilute it equals a certain percentage. One hundred grams of normal blood contains 14 grams of oxyhemoglobin.

Dare's Hemoglobinometer.—This instrument (Fig. 21) is also based upon a color comparison. A tinted glass is compared with a definite thin film of blood (not diluted with water), candle-light being used for illumination.

Estimation of the Red Corpuscles.—Method of Counting the Blood-corpuscles by Means of the Thoma-Zeiss Apparatus.—This apparatus consists of a glass pipet containing a

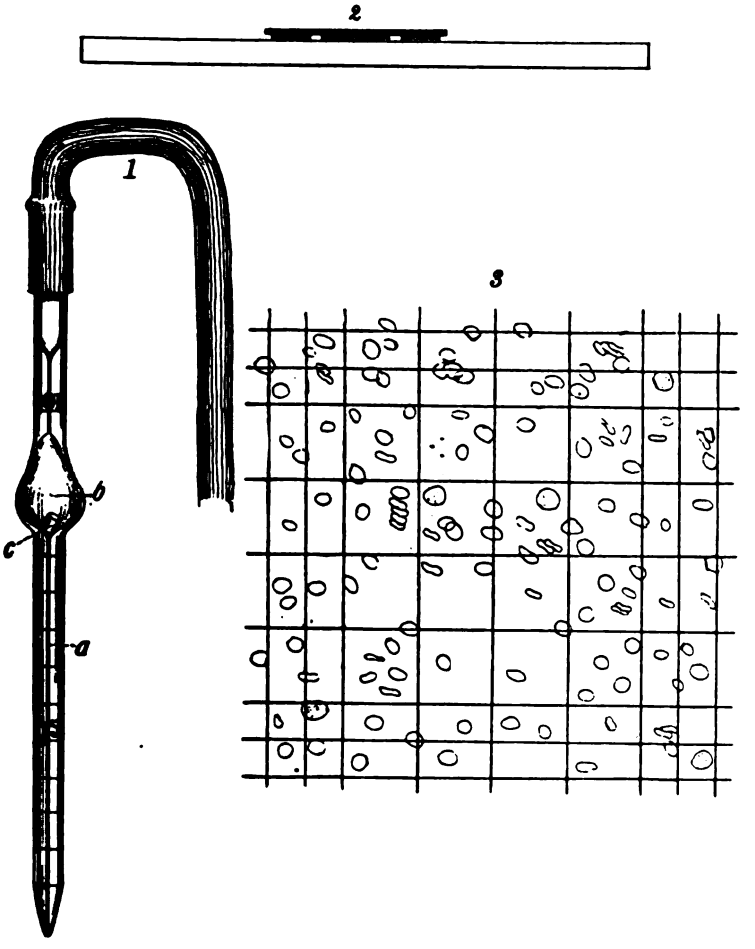


Fig. 22.—Thoma-Zeiss blood-counting apparatus. 1. Mixing apparatus: *a*, Capillary tube in which the blood is taken; *b*, chamber for mixing the blood with the diluting solution; *c*, glass ball to aid in mixing the blood with the diluting solution. 2. Cross-section of the chamber in which the blood is counted. 3. Section of the field on which the blood is counted, showing thirty-six squares.

capillary tube expanding into a bulb in which lies a glass ball; the bulb is then contracted and again merges into a small

capillary portion. The lower end of the capillary bore is graduated into 10 parts—from $\frac{1}{10}$ to 1. The capillary portion above the bulb is marked 101.

By filling the pipet with blood up to the point 1, and then with some diluting solution up to the point 101, a solution of 1 : 100 is obtained. By filling the pipet up to the point $\frac{5}{10}$, and the diluting solution up to the point 101, a solution of 1 : 200 is obtained. The other part of the instrument is the counting chamber. This consists of a thick glass slide upon which is cemented a disc, upon the surface of which are a number of rulings dividing it into 400 squares, each of these squares measuring $\frac{1}{20}$ of a millimeter in length and the same in breadth. Around this disc there is a depression formed by a hollowed cover-glass cemented upon the slide. This glass plate is $\frac{1}{10}$ of a millimeter thicker than the disc. When a cover-glass is placed upon the counting chamber, the space overlying each square is $\frac{1}{4000}$ of a cubic millimeter. Double rulings are also found upon the disc, these being so arranged as to indicate or outline 16 squares. Certain solutions are recommended for diluting the blood. Toisson's solution gives very good results; it is of special value, as it stains the white corpuscles a violet or blue color.

The formula is as follows:

Methyl-violet, 5 B	0.025 gm.
Chlorid of sodium	1.000 "
Sulphate of sodium	8.000 "
Neutral glycerin	30.000 c.c.
Distilled water	160.000 "

Gowers' diluting fluid also gives good results, the formula being as follows:

Sulphate of sodium	gr. cxij
Acetic acid	℥v
Water	℥iv.

A 2½% solution of bichromate of potassium is strongly recommended by some authorities, and gives excellent results. A 3% sodium chlorid solution may also be employed for the same purpose.

Method of Procedure.—The pipet must be perfectly clean. The usual precautions of obtaining the blood are to be observed. The blood is now sucked into the pipet up to the point of $\frac{5}{10}$ or 1. When great anemia exists, it is always best to fill the pipet up to the point 1. In cases when the blood

is near the normal, a dilution of 1 : 200 should be used—that is, the pipet is filled to the point $\frac{5}{10}$. Care should be taken to wipe off the point of the pipet before placing it into the diluting fluid, which is sucked up to the point 101, and in doing so the pipet is gently tapped so as to mix the blood and prevent it from floating upon the top. The diluting solution must always be filtered before using. The pipet is now shaken so as to thoroughly mix the blood and the diluting fluid, and the fluid in the capillary portions is expelled. A small drop of the mixture is placed upon the counting stage and the cover-glass adjusted; when this has been done, we should be able to observe "Newton's rings" beneath the cover-glass and the top of the instrument. In adjusting the cover-glass care must be taken not to get any of the fluid or particles of dust beneath the cover-glass that is in relation to the top of the counting-chamber. The counting-stage is then carefully placed upon the stage of the microscope, caution being exercised to have it perfectly level. A fourth or fifth objective is best suited for counting. The number of corpuscles overlying a given number of squares is now enumerated. If the distribution of corpuscles be uniform, 16 squares will be sufficient, otherwise a greater number should be used. The greater the number of squares employed in counting, the more accurate the result. Corpuscles found in relation to the upper and right-hand boundary line of a square are enumerated with it. In counting it is best to begin at the upper left-hand corner, 16 squares, counting from left to right until 4 squares have been used, and then from right to left in the lower layer, and so on in this serpentine manner.

The method of calculating the number of corpuscles per cubic millimeter is as follows: The number of corpuscles counted is multiplied by 4000 and this by the degree of dilution, and the product divided by the number of squares employed, the result being the number per cubic millimeter.

Formula:

$$\frac{\text{Number of corpuscles counted} \times 4000 \times \text{degree of dilution}}{\text{Number of squares used in counting}} = \text{Number of corpuscles per cubic millimeter.}$$

Carefully cleanse the pipet after using. After blowing out the solution it should be washed with a weak solution of acetic acid if Toisson's solution has been used; if other diluting solutions are employed, it is rinsed with water; next, it should be washed with alcohol and, finally, with ether; a column of

air being then passed through so as to thoroughly dry the instrument. It will be found laborious to fill and empty the tube. A pump or atomizer bulb may be used for the purpose of blowing out the fluid. A much easier method is to have a rather firm piece of rubber tube attached to the pipet, which, when twisted and the end firmly compressed between the fingers, will be found to drive the fluid from the tube. If the tube be placed in a fluid and the rubber tube straightened by untwisting, a vacuum is created, thus causing the fluid to flow into the tube. After having rinsed the tube with ether or alcohol it is best dried by holding it some distance from a flame,—not allowing the flame to touch the glass,—and then by a series of twists as just described the air rushes in and out, and the apparatus is thoroughly dried in a few minutes.

Enumeration of White Corpuscles.—In order to more accurately enumerate these corpuscles, a pipet having a capillary portion of larger bore than that for counting the red blood-corpuscles is used, so that a dilution of 1 : 10 is secured. The fluid used for this purpose is a watery solution of acetic acid, the strength of which is from ½ % to 1 %. This serves to dissolve the erythrocytes, rendering the white corpuscles more plainly visible. The method of counting is similar to that described for enumerating the erythrocytes, except that for the white corpuscles a stronger dilution, 1 : 10 or 1 : 20, is employed. Other methods have been used for counting the corpuscles, such as by the hematokrit, the Oliver tintometer, Gowers' hemocytometer, and by Durham's modified hemocytometer, descriptions of which will be found in special works upon the blood.

Blood Staining.—In order to study the more minute structure of the blood it is necessary to stain it; before doing this, however, a cover-slip preparation must be fixed. Cover-glass specimens are prepared by taking a thin cover-glass (which should be scrupulously clean), and allowing it to touch a drop of blood without coming in contact with the skin. This is permitted to fall upon a clean cover-glass, the two cover-glasses being rapidly drawn apart without lifting. The specimens are fixed preferably by heating to a temperature of from 130° to 150° C., for from fifteen to thirty minutes, in an oven or upon a copper plate. This method of fixation is always employed when using Ehrlich's stain. The covers may also be fixed by immersion in equal parts of absolute alcohol and ether, being immersed for half an hour. Other methods of

fixation are sometimes employed, such as solutions of formaldehyd or absolute alcohol.

Staining.—The method most often employed is the one proposed by Ehrlich. This stain consists of—

Saturated aqueous solution of orange G.	6 parts
Saturated aqueous solution of acid fuchsin	4 "

These solutions are mixed, and then is added—

Saturated aqueous solution of methyl-green . . .	6.6 parts.

To this mixture is added—

Glycerin (neutral)	5 parts.
Absolute alcohol	10 "
Water	15 "

The mixture should be shaken and allowed to stand for twenty-four hours. It must not be filtered. The cover-glass spreads are stained for five minutes with Ehrlich's tricolored mixture, then washed with water, dried well, and mounted in Canada balsam. To intensify the nuclear stain after employing Ehrlich's mixture, it is advisable to stain the specimen for ten or twenty seconds with a saturated aqueous solution of methyl-green. For differential counting it is essential to use this stain; but for the demonstration of nuclei or malarial parasites, a double stain, using a more intense nuclear dye, is preferable. The specimen should then be fixed in alcohol and ether. The stain ordinarily employed consists of the following procedure: Stain the specimen with a solution consisting of ½% of eosin, 50 parts of alcohol, and 50 parts of water, for from three to five minutes. Wash the stain off with water, and, finally, stain with some nuclear dye, such as a solution of hematoxylin, methylene-blue, or toluidin-blue, for from two to five minutes. Wash with water, dry, and mount with Canada balsam.

Description of the Erythrocytes.—The number of red cells per cubic millimeter in health is 5,000,000 in an adult male, and 4,500,000 in an adult female. This number varies somewhat under normal conditions: In strong, full-blooded individuals 6,000,000 corpuscles are sometimes seen. An increase in the number of red cells is termed *polycythemia*, which may be met with, as just mentioned, in vigorous individuals, in early childhood, in high altitudes, in cyanosis, and from blood concentration, the latter being produced after severe diarrhea, vomiting, or excessive sweating; hence, this

is met with in cholera, and at the crisis in some of the acute infectious diseases.

A decrease in the number of erythrocytes is termed *oligocythemia*. This condition is encountered as a result of menstruation, pregnancy, childbirth, lactation, after meals, fatigue, and direct blood loss, and in the primary and secondary anemias; the greatest reduction is found in pernicious anemia.

The red blood-cell (erythrocyte) is a biconcave disc measuring about 7.5 microns in diameter. The size, however, varies somewhat under normal conditions: 75% of the corpuscles measuring exactly 7.5 microns in diameter, while 25% vary from 6 to 9 microns in diameter. The erythrocyte is of a yellowish-green color, and consists of a delicate stroma—called the stroma of Rollett—in the meshes of which is held the hemoglobin as an albuminate, which is soluble in water. It stains with the acid dyes; with Ehrlich's triple stain it has a selective affinity for the aurantia. The tendency to rouleaux formation and crenation occurs normally. Its absence is abnormal. The size of the red corpuscles varies under pathologic conditions; a small corpuscle is called a *microcyte*. When a number of corpuscles are below the average size, the condition is termed *microcytosis*. This is commonly met with in chlorosis and in some of the secondary anemias. In this condition the percentage of hemoglobin is lower than the percentage of the corpuscles. A large red corpuscle is called a *macrocyte;* and when a number of corpuscles are found larger than normal the condition is called *macrocytosis*. This condition is frequently met with in progressive pernicious anemia, in which affection the percentage of hemoglobin is greater than the percentage of red corpuscles. An erythrocyte having a distorted outline is called a *poikilocyte*. When a number of the erythrocytes present this appearance, the condition is known as *poikilocytosis*. The shapes assumed by the corpuscles vary greatly—they may be pear-shaped, dumb-bell-shaped, etc. This condition may be due to artifacts. When this is the cause, the long diameters of the distorted corpuscles are almost in parallel lines, and is caused by the sliding apart of the cover-glass in making the spreads. Pathologic poikilocytosis occurs in all severe anemias, especially in progressive pernicious anemia.

Nucleated red corpuscles are found normally in the blood of the embryo up to the seventh month; occasionally, they may be found at birth. The varieties of nucleated cells are

the *normoblasts*, the *megaloblasts*, the *microblasts*, and the *poikiloblasts*.

The *normoblast* is about the size of the normal erythrocyte, and has a nucleus that occupies one-third or one-half of the cell. When stained with Ehrlich's triple mixture, the perinuclear protoplasm stains a faint or light-yellow color, while the nucleus is colored a dark-green or blue. Occasionally, it is found that there appears about the nucleus a faint hyaline or clear space denoting a separation between the nucleus and the perinuclear protoplasm. The nucleus may occupy the center or the periphery of the cell, in some instances extruding from the cell. It may present variations as to the staining reaction, being darker in some places and lighter in others; and, rarely, there are mitotic figures. This cell is normally found in the bone-marrow, and probably represents an early stage in the development of the erythrocyte. After copious hemorrhage these cells are found in the circulating blood, and it appears that they are thrown into the circulation before the matured cell is developed. They are also found in great numbers in splenomedullary leukemia, the bone-marrow in this disease revealing marked proliferative changes.

The *megaloblast* is a very large nucleated red cell, its size varying from ten to twenty microns in diameter. It has a large nucleus, which possesses a weak selective affinity for the basic stains. When treated with Ehrlich's tricolored mixture, it is colored a robin's-egg blue, the stain being either uniformly distributed or, in some instances, irregularly. Around this nucleus there appears an unstained area, it being separated from the perinuclear protoplasm, the latter often revealing degenerative changes, so that it does not take up the acid stain properly, and is of a brownish-red color. The megaloblast probably represents a fetal type of cell development. It is never found in normal bone-marrow in the adult, but in fetal marrow, and in the blood and marrow of some of the grave forms of anemias.

The *microblast* is a small cell containing a deep-staining nucleus; it is less frequently found than the cells just described.

When the red blood-cell is distorted and contains a nucleus it is called a *poikiloblast*. This cell must be regarded as having the same clinical significance as the megaloblast, and, like that cell, often shows degenerative changes in the perinuclear protoplasm.

Double and triple nucleated red blood-cells are sometimes encountered, notably in splenomedullary leukemia.

The normal erythrocyte possesses the power of selecting acid dyes, such as eosin, acid fuchsin, picric acid, and aurantium. Under certain conditions the staining reaction is altered, so that the cell will take up not only acid dyes, but a mixture of acid and basic dyes, and under such circumstances, when stained with Ehrlich's triple mixture, the cell is of a brownish-yellow or brownish-red color. When these changes are encountered, they should be regarded as evidences of degeneration (polychromatophilic red corpuscles).

In the stained preparations faint rings are sometimes noticed, the protoplasm of the same having been washed out. These cells are known as *shadow* or *phantom corpuscles*.

The Hemoglobin.—In the normal state the hemoglobin is contained in the red blood-cell as a soluble albuminate; a deficiency in the hemoglobin indicates a loss of albumin. Every 100 grams of blood represent 14 grams of hemoglobin, which is expressed as being 100%. In all forms of anemia the hemoglobin is decreased. When the percentage of hemoglobin and the percentage of erythrocytes are equal, it may be inferred that each red blood-cell possesses a correct amount of coloring-matter, this being termed the *color index*, or corpuscular richness, in hemoglobin. When the erythrocyte possesses the normal amount of coloring-matter, it is said that the color index is normal, or equals 1; when the cell contains less hemoglobin than normal, it is said that the color index is low; when it contains an excess, we speak of the color index as high, or above 1. The exact index is determined by dividing the percentage of hemoglobin by the percentage of red corpuscles; for example, if the number of red cells is 5,000,000 per cubic millimeter, or 100%, and the hemoglobin be 50%, the color index will be 0.5. The hemoglobin is more difficultly regenerated than the red blood-corpuscles, so that in nearly all forms of anemia the color index is low, except in progressive pernicious anemia, when it is usually high.

The Leukocytes.—In the healthy adult there are from 6000 to 8000 white blood-cells per cubic millimeter in the peripheral circulation. Increase in the number of leukocytes is termed *leukocytosis*. This may be either physiologic or pathologic. A physiologic leukocytosis is encountered during pregnancy, during digestion, in the new-born, and just

before death, the latter being called *agonal* or *terminal leukocytosis*. It is also encountered after exercise, bathing, and massage. Pathologic leukocytosis may be due to inflammatory or infectious causes, when it is termed *inflammatory leukocytosis;* or after hemorrhage, when it is called *posthemorrhagic leukocytosis*. Leukocytosis is met with during the course of sarcoma and carcinoma, known as *malignant* leukocytosis; when resulting from toxic changes, it is called *toxic leukocytosis*.

Inflammatory leukocytosis is met with in nearly all inflammations and infectious diseases, except enteric fever, tuberculosis, malaria, influenza, measles, and rötheln. When the resisting powers of the individual are good, a slight leukocytosis is encountered from mild infections; and a marked leukocytosis, from severe infections. When the resisting powers of the individual are feeble, no leukocytosis is encountered from very severe infections. It is said that with overwhelming infections even with good resisting powers there is no increase in the number of white blood-cells. After copious hemorrhages a leukocytosis of from 10,000 to 20,000 is met with. Malignant leukocytosis is encountered during the growth of sarcoma and carcinoma; the former, as a rule, revealing a more marked leukocytosis than the latter, so that a leukocytosis of from 20,000 to 50,000 rather indicates sarcoma than carcinoma.

Leukopenia (Hypoleukocytosis).—These terms are used to express a decrease in the number of white blood-cells in the peripheral circulation. It is met with in the following conditions: starvation and prolonged cold bathing; and sometimes in tuberculosis, malaria, enteric fever, and pernicious anemia.

Varieties of Leukocytes.—*1. Polymorphonuclear Neutrophile.*—This cell is sometimes termed the *polynuclear leukocyte* or the *finely granular oxyphilic cell.* It measures about 13½ microns in diameter, is ameboid, and phagocytic. It possesses a nucleus that is irregular in shape, lobulated or twisted, and often appears as though there were many of them; hence the name *polynuclear leukocyte.* When the cell is stained with strong basic dyes, it will be found that there is always a single nucleus, the lobulated portions being joined by chromatin bands. When stained with Ehrlich's triple mixture, reddish-brown or purple granules are seen in the portion of the cell surrounding the nucleus. These granules possess a marked

affinity for the acid stains, so that the term "finely gr
oxyphilic cell" seems to be more properly applied.
the most frequent leukocyte encountered in normal
it forms from 60% to 70% of all the leukocytes.

2. The Eosinophile, Polynuclear Eosinophile, or Coarsely ular Oxyphilic Cell.—This white blood-corpuscle me about twelve microns in diameter; it is ameboid, but is sa to be phagocytic; it possesses a nucleus similar to that s the polynuclear neutrophile. In the perinuclear protoplas seen granules that are large and spheric in outline; thes a strong affinity for acid dyes, and when stained with appear as bright-red bodies; hence the term "eosinop They form from ½% to 4% of the leukocytes in n blood. When they occur in large numbers, the term *eosino* is employed. This condition is met with in asthma, in of the diseases of the skin, and in trichinosis. Under n conditions they are found in large numbers in the serous ities.

3. The Small Lymphocyte or Small Mononuclear Cell (. times called the Small Hyaline Cell).—This cell is the sm of the leukocytes, its average diameter being about microns. It possesses a single large nucleus that occ the greater portion of the cell, and is surrounded by a rim of protoplasm. The nucleus reacts to the basic dyes marked affinity, so that when it is stained with Ehrlich's ture it appears blue. The protoplasm reacts faintly to the dyes, and with the triple mixture stains a light pink. Somet it stains a pale-blue color. This is met with when the b stain is in excess. The small lymphocytes comprise f 20% to 30% of all the leukocytes in normal blood; t are, however, found in large numbers in the lymphatic ch nels. In some diseases the lymphocytes are increased; thi met with in lymphatic leukemia and progressive pernici anemia. The cell does not possess ameboid or phagoc properties.

4. The Large Lymphocyte, Large Mononuclear or Hya Cell.—The size of this leukocyte is about thirteen micron diameter. It is ameboid and phagocytic. Its morphol closely resembles that of the small lymphocyte, except t the cell is larger; the nucleus, however, is not so large proportion to the size of the cell, and has a weaker affinity the basic dyes. It comprises from 4% to 8% of the leu cytes.

5. The Mast-cell or Coarsely Granular Basophilic Leukocyte.—This cell is said to occur in normal blood in the proportion of from $\frac{1}{10}\%$ to $\frac{1}{2}\%$. It is found more frequently in the blood of certain forms of leukemia. It possesses a nucleus that is twisted or lobed, and in the perinuclear protoplasm there are found very coarse granules that react to basic stains. Mast-cells are found abundantly in some connective tissues; they are described as having granules, possessing a modified basophilic reaction—that is, when they are stained with toluidin-blue and treated with glycerin-ether or a weak oxalic acid solution, the color of the stain is modified from blue to purple or red. The five varieties of leukocytes just described are found in normal blood.

Under certain pathologic changes another cell, called the myelocyte, may be found.

The Myelocyte (Marrow Cell or Mark Cell).—This cell is normally found in the bone-marrow. As a rule, it is quite large, the average diameter being about 15.75 microns; the variation in size is from 10 to 20 microns. It may be described as having a large, pale-staining nucleus, the contour of which varies from the round oval to the lobe shaped. There is a faint rim of protoplasm around this nucleus, which contains granules. They may be fine and react to the acid stains (hence the name neutrophilic myelocyte or finely granular oxyphilic myelocyte); the granules may be coarse and react to the acid stains (eosinophilic myelocytes or coarsely granular oxyphilic myelocytes); or, in some rare instances, they may contain fine granules which react to the basic stain.

The myelocyte is easily differentiated from the large lymphocyte in that the latter does not contain granules. It is also easily differentiated from the polynuclear neutrophile in that the former possesses a large, pale-staining nucleus. The nucleus of the myelocyte sometimes reveals an irregular staining reaction, and, occasionally, vacuoles are noted. This cell, as above stated, only appears in the circulating blood under morbid conditions, it being found in large numbers in splenomedullary leukemia.

Blood Plates.—These bodies are found in the blood as irregularly shaped masses about one-half the size of the erythrocyte, and are usually found in clumps or large masses. They are rather difficult to demonstrate in a fresh specimen of blood, and appear as colorless plates. A cubic millimeter of normal blood contains from 20,000 to 700,000 of these

bodies. From a clinical standpoint they are of l
ance. A decrease in the number is met with in
hemophilia. They are increased in leukemia.

Müller's Blood Dust (Hemokonien).—In norma
mal blood small hyaline refractive bodies, possessir
molecular motion, are frequently seen. Their s
constant, frequently appearing as dumb-bell ma
are of no clinical significance.

Chemic Examination of the Blood.—The cl
of the chemic examination of the blood at the p
is of little practical significance, since it is not well
the technic being also lacking.

A very small amount of grape-sugar is present
blood, which is increased in diabetes mellitus.
minute quantities of uric acid are found in the bl
jaundice bile will be found. The methods for cli
mination of these substances have not been per
ciently to be of any practical clinical value.

When fat is found in the blood, the condition
lipemia. This is met with in diabetes mellitus,
alcoholism, and in chronic nephritis.

When acetone is found in the blood the conditio
acetonemia. It occurs in fevers, and particularly i

When excrementitious substances that are parti
rived from urinary products collect in the blood,
condition arises called *uremia*.

The most common inorganic constituent of th
sodium chlorid, which is found in the proportion of

Micro-organisms Found in the Blood.—The mo
of these is the spirillum of relapsing fever, which is
in the chapter on Bacteriology. The bacillus of anth
culosis, glanders, bacillus typhosus, influenza, and
munis are rarely found in the blood. The staphyloc
the streptococcus have also been described as occur
blood. (For description of these micro-organisms, s
on Bacteriology.)

Animal Parasites Found in the Blood.—The pl
malariæ is the most common animal parasite met w
blood. (For description, see page 198.) The distor
tobium and filaria sanguinis hominis are blood
(For description, see section on Parasites.)

Description for Plate I.

Figures 1 to 35, inclusive, stained with Ehrlich's triple mixture to 51, inclusive, stained with eosin and toluidin-blue, as recommend

Figs. 1, 2, 3.—Polymorphonuclear neutrophiles.

Figs. 4, 5, 6.—Eosinophiles.

Fig. 8.—Small lymphocyte.

Fig. 12.—Large lymphocyte.

Figs. 7, 9.—Small lymphocytes (from the blood in lymphatic leuke atypical staining reaction).

Figs. 10, 11.—Large lymphocytes (also showing atypical staining)

Figs. 13, 14.—Myelocytes (with neutrophilic granules).

Fig. 15.—Myelocyte (with eosinophilic granules).

Figs. 16, 17.—Erythrocytes.

Figs. 18, 19.—Erythrocytes showing normal variation in size.

Figs. 20, 21.—Macrocytes.

Figs. 22, 23.—Microcytes.

Figs. 24, 25, 26, 27, 28.—Poikilocytes.

Figs. 29, 30.—Normoblasts.

Figs. 31, 32.—Red blood-cells, showing irregularly shaped nuclei.

Fig. 33.—Megaloblast.

Fig. 34.—Microblast.

Fig. 35.—Erythrocyte, showing degenerative changes as demonst polychromatophilic reaction.

Figs. 36, 37, 38.—Polymorphonuclear leukocytes.

Figs. 39, 40.—Eosinophiles.

Figs. 41, 42, 43.—Small lymphocytes.

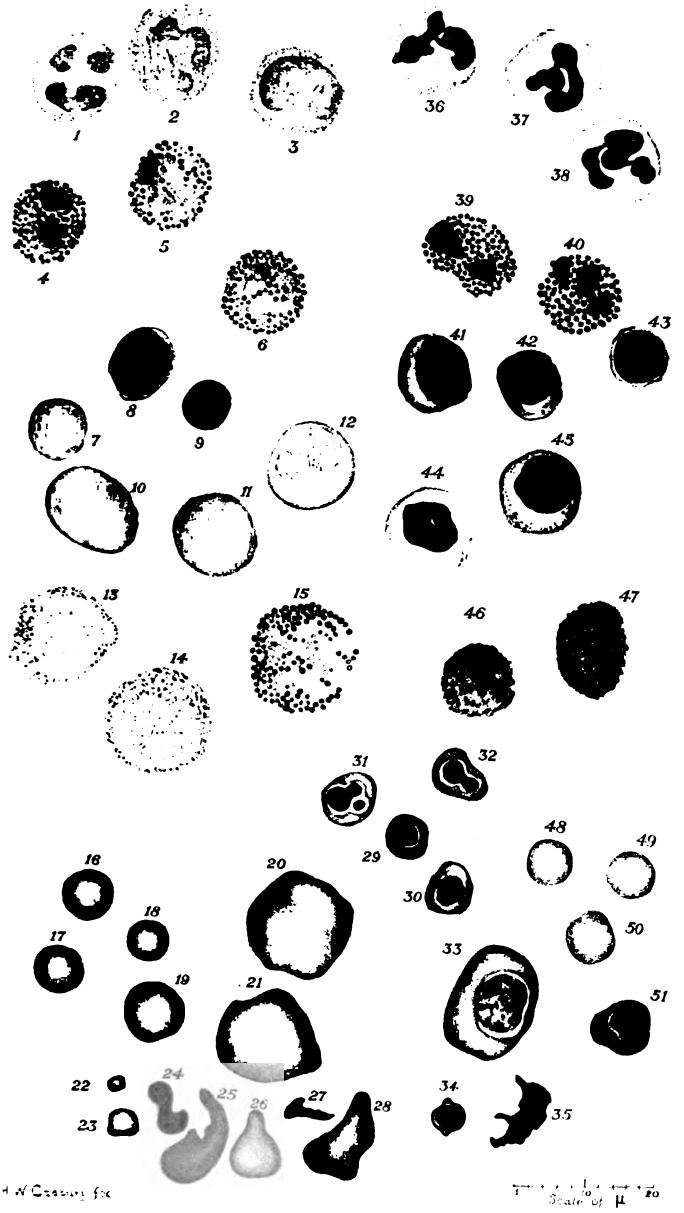
Figs. 44, 45.—Large lymphocytes.

Figs. 46, 47.—Myelocytes.

Figs. 48, 49, 50.—Erythrocytes.

Fig. 51.—Normoblast.

PLATE I.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EXAMINATION OF THE URINE.

The normal quantity of urine in twenty-four hours is 1500 c.c.; however, this amount varies greatly, depending upon many physiologic and pathologic changes. The amount of urine shows diurnal variation—the greatest activity in the flow being during the day; this is less at night and in the early morning. The amount is increased after partaking of fluid, anxiety, or excitement.

The urine is light amber or a pale straw color, varying to a reddish-yellow. When it becomes concentrated, the shade becomes darker; and when diluted, it is lighter. The pigments found in the urine that are of the most importance are urobilin and indican. The urine is perfectly clear and transparent, but upon standing a slight sediment or cloudiness is noticed, this being due to mucus. It occurs in nearly all forms of normal urine. Sometimes under normal conditions, after it becomes cool, uric acid salts deposit.

The specific gravity varies between 1015 and 1020, depending on the degree of concentration and the amount of solids, which vary somewhat with physiologic conditions. The specific gravity is determined by the use of the ordinary urinometer. The amount of solids that are found in twenty-four hours in normal urine ranges from 60 to 70 grams.

During the course of twenty-four hours the reaction of the urine varies somewhat, but it is always acid. With a mixed diet the acidity decreases, but some hours after a meal the reaction again becomes decidedly acid. The urine may give an amphoteric reaction; by this is meant that red litmus is rendered blue, and blue litmus red.

It has a faint aromatic odor, but this varies, depending largely upon the diet. After eating asparagus it emits a stench; after onions or garlic there is a garlicky odor; and after partaking of certain drugs, such as turpentine, there is an odor resembling that of violets. Copaiba, cubebs, asafetida, musk, valerian, and castor oil produce a faint odor similar to that produced by turpentine.

Micro-organisms are found in normal urine, such as the bacteria urea and the micrococcus urea, often the smegma bacillus.

The amount of urea passed in twenty-four hours by a healthy adult is from 25 to 40 grams. The amount of uric acid passed in twenty-four hours is 0.66 gram. This

varies within wide limits. The amount of sodium
passed in twenty-four hours is from 10 to 16 grams
faint traces of bile pigments are noted in normal urin

Alterations in the Amount Secreted.—An increa
amount of urine is called *polyuria*. It may result fro
creased arterial pressure, arising from the partaking c
and some drugs, as digitalis, caffein, etc.; it may
from nervous causes, as in hysteric individuals; also
betes mellitus and insipidus; in chronic interstitial
tis; diseases of the brain, such as irritation of the floor
fourth ventricle; from hemorrhage into certain parts
brain; and from the partaking of large quantities of l
as beer, wine, and coffee; also from the resorption of
and transudates. During the convalescence of some
fevers, and particularly during the crisis (critical discha
there is an increase in the amount of the urine, and fin
sudden increase is noted temporarily, resulting from the
of an obstruction to some part of the conducting appa
as from the plugging up of the ureter by stone or from
modic contraction of the urethra, etc.

A decrease in the quantity of urine is called *oliguria*.
is caused by a decrease in the blood pressure, which oc
from failing heart compensation; from slowing of the circ
tion through the kidney, as from embolus in the renal arte
also from increased sweating or diarrhea, as is common
choleric diseases; and from obstinate vomiting. During
course of the fevers the amount is decreased. It may be
to nervous causes, such as shock; also in disease of t
kidney, such as acute and chronic parenchymatous nephrit
Diseases of the conducting apparatus may cause a dammi
back of the flow of the urine, as is common in stricture
the urethra. In uremia the flow of urine is always diminishe
and sometimes completely absent. After severe hemorrhage
particularly internal hemorrhages, the amount of urine is de
creased. This depends upon the lowering of the arteria
pressure. After the removal of a kidney the amount may b
temporarily decreased, the other kidney from reflex actior
failing to secrete urine.

Color.—Various diseases produce changes in the color of
the urine. Blood in the urine (hematuria) is caused by the
presence of red blood-corpuscles in the urine, the color being
of a reddish hue. If free hemoglobin be found, it is called
hemoglobinuria. In this condition the urine is of a reddish

color, varying to a brownish-black, the intensity of the color depending upon the amount of blood pigment present. When blood is derived from the bladder, the first that is passed will contain a smaller amount of blood than the last. Blood from the bladder may be copious in amount, and this urine, in contrast to that containing blood from the kidney, upon standing will show fibrin. Blood from the kidney is more likely to be intimately mixed with the urine, and if coagula be present, they are apt to be washed out. From the urethra, only the last drops are likely to be bloody. Hemoglobinuria occurs from a number of causes, the most important being malaria, variola, purpura, scurvy, thermic fever, and large burns of the skin; also from certain drugs, as chlorid of potassium, pyrogallic acid, quinin, or carbolic acid. When bile is mixed with the urine, as occurs in various forms of jaundice, the color becomes dark yellow, yellowish-green, brownish, or even porter color. The foam upon the urine is retained for some time.

In *chyluria* or *galacturia* the urine has a milky or chylous appearance. If it is allowed to stand, a fatty deposit will appear upon the surface, and upon microscopic examination of this material fat is observed, which is dissolved by ether or alcohol. Chyluria is principally met with in the tropics, and is due particularly to the filiaria sanguinis hominis. The urine may slightly change in color as the result of fat, which occurs from the following causes: In diseases of the kidney, such as chronic parenchymatous nephritis, pyonephrosis, or spermatorrhea; in the severe cachexias, such as tuberculosis, yellow fever, pyemia, or poisoning from coal-gas and phosphorus. *Melanuria* is due to melanin being present in the urine. This gives rise to a brownish or blackish discoloration after the urine has been standing, even if it was passed clear. This condition takes place in melanotic tumors, particularly sarcoma. *Indicanuria* gives rise to a bluish or bluish-black appearance of the urine, and results from the following conditions: The accumulation of feces from obstinate constipation, from peritonitis, from severe cachexias, and from intestinal catarrh. The color of the urine is often influenced by drugs: from methylene-blue, the urine becomes blue; from carbolic acid, creosote, salol, and the coal-tar products, it becomes greenish-black; from hematoxylin or log-wood the color becomes reddish or purplish; from santonin and picric acid, it becomes yellowish-green in color; and from juniper

berries, rhubarb, senna, and chrysarobin, it becom
red. Cloudiness or turbidity may result from an
mucus, from phosphates, urates, blood, pus, chyle
cells, and numerous bacteria.

The specific gravity of the urine in disease va
wide limits, from somewhat over 1000 to 1074. Hi
gravity is found in the following diseases: in fe
failure of heart compensation, acute and chronic par
tous nephritis. A very high specific gravity of
occurs in sulphuric acid poisoning. It also often ta
after the use of certain remedies, such as diuretics
potassium acetate, and different tartrates. Very hig
gravity is met with in diabetes mellitus; low specific
encountered in the following diseases: chronic i
nephritis and diabetes insipidus. The specific gravity
as low as 1002.

Reaction of the Urine in Disease.—Neutral or
reaction of the urine may result from the ingestion of
(except ammonia), from the resorption of large transud
exudates, or hemorrhages, or from free blood in the ur
sometimes after hot and cold baths. The urine may be
as the result of gastrectasis. This is especially so if
panied by frequent vomiting. It may also result from f
tation of the urine in the bladder, particularly when mixe
large amounts of pus.

Acidity of the Urine.—Hyperacidity is of very little
nostic importance. For practical clinical purposes the re
of the urine is determined by litmus paper.

Chemic Examination of the Urine.—The occurren
albumin in the urine is called *albuminuria*. In the majori
cases it is either serum albumin or seroglobulin (paraglob
Albumin results from diseases of the kidney, in fevers,
acute poisoning. It may also result from the admixtur
blood or pus.

True or renal albuminuria is due to diseases of
kidneys themselves, while *toxic albumin* appears as the re
of toxemia.

Test for Albumin.—When the urine is cloudy, it sho
be filtered, and the filtrate tested. Mucin-like substances m
resemble the reaction for albumin. This is best avoided
acidulating the urine with acetic acid and filtering. Repeat
filtrations are sometimes necessary.

Heat and Acid Test.—The urine is placed in a test-tube, ar

boiled; if albumin be present, a white precipitate will be noted. If cloudiness result after boiling, it may be due to phosphates, but on the addition of acid (nitric or acetic) the phosphates will clear up, and albumin will be precipitated.

Heller's Test.—This is performed by placing a quantity of nitric acid at the bottom of a test-tube and allowing the urine to flow gently down the side of the tube, so that the urine overlies the nitric acid; if albumin be present, a white disc is formed at the point of contact. If urates be present, a yellowish-white disc may appear at the junction of the two liquids. This is often misleading. Upon the gentle application of heat the urates disappear and the albumin remains.

The Test by Acetic Acid and Potassium Ferrocyanid.—The method suggested by v. Jacksch is as follows: Acetic acid and a few drops of a solution of ferrocyanid of potassium are placed in a test-tube, and the urine is superimposed upon this mixture. If albumin be present, a white disc will appear at the line of contact. This is a very delicate test.

The Picric Acid Test.—This test is best accomplished by placing urine in a test-tube and allowing a saturated solution of picric acid to flow gently into the tube; if albumin be present, a white disc will form at the line of contact, and at this point a diminished transparency is noted. If the patient has been taking quinin, thallin, antipyrin, or potassium salts, the picric acid test will be open to fallacies, as thick precipitates may appear that are not albumin. It is also obtained by the presence of mucin.

Fig. 23.—Esbach's albuminometer.

Quantitative Test.—The Esbach albuminometer (Fig. 23) consists of a somewhat thickened test-tube, which is graduated and divided into eight lines beginning at ½ and then going to 7. There are two divisions, one marked "U" and the uppermost marked "R." The urine is poured into the tube to the point marked "U" (urine), and the reagent to the mark "R." The reagent fluid consists of 10 grams of picric acid, 20 grams of

citric acid, and 1000 grams of water. The tube is t
with a rubber cork, shaken, and allowed to stand
four hours. If the precipitate should reach to the
this would signify that 1000 c.c. of urine contain
albumin, or a 0.2%. Each mark represents $\frac{1}{10}$
amount should reach to the figure 7, the urine must
one-half, and the result multiplied by 2. The roo
the test is made should not have too low a tempe
urines that contain less than 0.2% of albumin, th
is spoken of as slight albuminuria; those in which t
shows 0.8% are known as medium albuminuria;
his would be designated as great albuminuria. It is
in performing this test to take a sample of the tot
collected during twenty-four hours.

Test for Bile Pigments and Bile Acids.—*Marèc*
—If a few drops of an iodin solution (Lugol's s
added to urine containing bile pigment, a grass-g
rapidly develops, revealing the presence of bile pign

Roscubach's Modification of Gmelin's Test.—Th
filtered; the filter is unfolded, placed upon a porce
and upon it a few drops of a mixture of nitric a
acids are poured. Color rings are produced—yello
violet, blue, and green—in which green predomina
color reaction shows the presence of bile pigment.

Grape-sugar.—The occurrence of grape-sugar in
is called *glycosuria*. In a number of pathologic
glucose is found in the urine, particularly in diabete
In this condition the amount of sugar is frequently
varying from 2% to 10%. The urine in this condition
increased in quantity, is of high specific gravity, and i
straw color, sometimes greenish. Glycosuria may
lesions of the floor of the fourth ventricle, such as t
cysts. It is met with in diseases of the liver, the
and occasionally results from the taking of large qu
starches or sugars; it is also sometimes met with
course of certain nervous conditions, such as epilepsy
apoplexy, injuries to the nervous system, neuralgia, a
sive mental exertion; also during pregnancy. It is
encountered during the course of the acute infectious
such as diphtheria, scarlet fever, cerebrospinal m
typhus fever, enteric fever, and cholera; from poiso
amyl nitrite, mercury, morphin, carbonic oxid, chlor
acid, sulphuric acid, and alcohol. Normal urine

slight trace of sugar, which is termed *physiologic glycosuria*. *Pathologic glycosuria* results from any of the morbid conditions just enumerated. This form may be transitory, when grape-sugar appears in the urine for a short time; or the sugar may be found in the urine constantly, when the condition receives the name of *persistent glycosuria*. The latter condition is met with in diabetes mellitus.

Tests for Grape-sugar.—*Trommer's Test.*—A small quantity of urine is added to one-fourth of its bulk of liquor potassæ, and to this mixture is added, drop by drop, a solution of cupric sulphate. The mixture is then boiled; if grape-sugar be present, a precipitate will form, which is yellowish or red in color, the substance being suboxid of copper. This test is quite delicate, and 0.001 part of sugar may be detected; however, some substances, when found in the urine, may give the reaction, such as uric acid, milk-sugar, kreatinin, bile pigments, chloral, and after the ingestion of benzoic and salicylic acid. If the urine contains large amounts of grape-sugar, the reduction takes place at a temperature below the boiling-point.

Fehling's Test.—This test may be substituted for Trommer's test. *Fehling's Solution:* This fluid must be freshly prepared before using, or the solutions can be kept separate and mixed when desired.

Solution (A): 34.639 grams of copper sulphate are dissolved in 100 grams of water by gently warming, and this is diluted to 500 grams.

Solution (B): To 173 grams of sodium tartrate are added 100 grams of a solution of sodium hydrate having a specific gravity of 1034, and this solution is dissolved in water until the mixture equals 500 grams.

To perform the test, equal parts of solutions A and B are mixed in a test-tube and diluted with four parts of water. This mixture is boiled; if the color remains blue, the solution has not spoiled. The urine is now poured into the test-tube, drop by drop, and gently boiled, and when sugar is present, the color changes to a yellowish or red color, the cupric sulphate being reduced. If upon boiling the color of the Fehling solution is not changed, and no precipitate forms, sugar is absent.

This solution may be used as a quantitative test, as it requires 0.05 gram of grape-sugar to reduce 10 c.c. of Fehling's solution. When using the quantitative method, the urine should always be diluted, the degree of dilution depending upon the amount of sugar present.

Fermentation Test.—Yeast decomposes grape-s
alcohol and carbonic acid. This test should alwa
ployed when reducing-substances are suspected in
which are not grape-sugar. The method is applied a
the test being that recommended by Dr. Robert
ounces of diabetic urine are placed in a twelve-oun
and compressed yeast is added to this; the bottle
lightly corked, so as to permit the carbonic acid to
This is set in a warm place. Another bottle containi
ounces of urine, tightly corked, is placed alongside
Fermentation will be complete in about twenty-four
After this time the specific gravity is taken of the fer
and the unfermented urine. It will be noted in the c
urine that after fermentation the specific gravity will b
ered. The number of degrees in specific gravity lost is
mined, and this result multiplied by 0.23; the resul
equal the percentage of sugar. The fermentation test is
delicate—0.1 % of sugar is detected in the urine. S
fermentation tubes are sometimes employed. The polari
is used to determine the presence of sugar.

Acetone.—Very small traces of this substance are pr
in normal urine, the condition being termed *physiologic ac
uria*. When it occurs in excessive amounts, it is ter
pathologic acetonuria. This substance is increased in fe
conditions, in diabetes, psychoses, derangements of digest
starvation, from auto-intoxications, carcinoma, and chlorof
narcosis. When acetone appears in diabetic urine, it indica
an advanced state of the disease.

Legal's Test.—Four cubic centimeters of urine are render
alkaline by the addition of liquor potassæ, and to this is add
a few drops of solution of sodium nitroprussid, a red col
being produced, which on the addition of acetic acid tur
purple if acetone be present.

Lieben's Test.—A small quantity of urine is distilled, th
distillate being used for the test, to which is added a few drop
of iodopotassic iodid solution and caustic potash. If aceton
be present, a precipitate of iodoform crystals is deposited,
which may be detected by its color and smell.

Diacetic Acid.—This substance is never found in normal
urine, and when present the condition is termed *diaceturia*. It
occurs in some auto-intoxications, in febrile conditions, and
diabetes. When diacetic acid is found in the urine, it is of

unfavorable diagnostic omen, and often indicates oncoming coma. Diacetic acid is accompanied by acetone.

Test.—The urine should be boiled, and a solution of chlorid of iron be added, drop by drop. If diacetic acid be present, a Burgundy red color is produced. It sometimes happens that phosphates are precipitated in performing the test. If this occurs, the phosphates should first be removed by filtration.

Ehrlich's Diazo Reaction.—The test is performed as follows: The reagents must be freshly prepared, and are—(1) Hydrochloric acid, 50 parts; distilled water, 1000 parts; and sulphanilic acid, 5 parts. (2) A 5% solution of sodium nitrite. Three grams of solution (1), with the addition of one drop of solution (2), are placed in a test-tube, and then an equal part of urine is added to this; mix carefully, and add about one-eighth of the volume of ammonia. If a red color develops upon shaking the mixture, the diazo reaction is present. This reaction is obtained from the urine in certain morbid states, particularly enteric fever, measles, and acute tuberculosis. Von Jaksch believes that the color when obtained is always due to the presence of acetone, and he prefers to regard the process rather as an uncertain indication of that body than as a test for anything else.

Urinary Sediments.—The urine is placed in a conic glass vessel, and allowed to stand for a number of hours so that the sediment will form in the pointed bottom, and then, by means of a pipet, it is withdrawn, placed upon a slide, and then a cover-glass is placed upon this. It is now ready for microscopic examination. The urinary sediment may be obtained by means of the centrifuge. When this instrument is available, the method is preferable, for when the urine is allowed to stand for any length of time changes develop, such as numerous micro-organisms.

Organized Sediments.—These consist mostly of various cellular bodies. **Epithelium.**—The normal urine always contains some epithelial cells. These are derived from the bladder, from the ureters, the renal pelvis, and sometimes from the substances of the kidneys. In inflammation of the mucous membrane of the genito-urinary tract large numbers of epithelial cells are present in the urine. In women, especially those suffering from leukorrhea, a number of large, flat epithelial cells are found in the urine. It is difficult to differentiate between the cells derived from the bladder, ureter, and renal

pelvis. Those cells which are derived from the superficial portion of the mucous membrane are polygonal and elliptic and contain a single large nucleus. The cells which are derived from the deeper layers are more irregularly oval in outline and also contain a single large nucleus. When the cells are in great abundance, they often indicate inflammation of the bladder. The clinical symptoms of the disease in question are necessary to discern between disease of the pelvis, the ureter, or the bladder. Cells which are derived from the tubules of the kidney are usually polyhedral in shape, and contain a large oval nucleus; but the shape of the cells varies, depending upon the portion of the urinary tubule from which they are derived. They may be found singly or coalesced, the latter indicating epithelial casts. The cells from the kidney often reveal degenerative changes—granular and fatty. The diagnosis of lesions of particular parts of the genito-urinary tract should never be based upon the shape of the epithelial cells alone, but other clinical manifestations of the disease in question must be taken into consideration.

Red Blood-cells.—Red blood-cells may occur in the urine without causing a change in color, and they may be discovered only by the aid of the microscope. They may be derived from any part of the genito-urinary tract. When these cells are intimately mixed with the urine, and upon microscopic examination appear as pale-yellow rings (phantom or shadow corpuscles), it may be inferred that their origin has been in the kidney, the pelvis, or the ureter, but a definite diagnosis should only be made if the other clinical facts coincide with the urinary findings. If the red blood-cells are not intimately mixed with the urine, and do not appear as washed-out corpuscles, it may indicate that their origin has been from the bladder or the urethra, but these facts should be combined with the other manifestations before an absolute diagnosis can be made.

Leukocytes.—Leukocytes in small numbers are found in the urine of healthy individuals, but when found in excessive amounts, indicate some disease of the genito-urinary tract. They may be derived from the urethra, bladder, ureter, pelvis, or from the kidney itself. When found in great numbers, they frequently indicate cystitis; when they are found as a result of inflammation of the pelvis or ureter, they appear in the urine in a considerable quantity, and, as a rule, in disease of the kidney substance itself a smaller number is found. Leukocytes

in the urinary sediment are quite easily recognized. The protoplasm of these cells frequently show degenerative changes; their outline may be quite irregular; the lobed nucleus is sometimes obscured, but on the addition of a small amount of acetic acid it can be seen. When pus is found in the urine, the condition is known as *pyuria;* when it is found in the urine in large amounts, it frequently deposits itself as a white sediment at the bottom of the vessel in which it is contained. This sediment often resembles that produced by amorphous phosphate of lime. Phosphates, however, are dissolved on the addition of acid, while pus is not.

Casts.—Tube-casts are said to be molds of the uriniferous tubules, produced either by substances in the lumen of the tubules without marked disease of the epithelial cells or by substance resulting from degeneration or coalescence of cells lining the tubules or contained in them. In the latter instance when formed from epithelium they are really not molds of the tubules themselves, but of their basement membrane. Casts are nearly always indicative of kidney lesion, but certain forms of them have been found in the urine of healthy individuals or after slight toxic influences. They are usually found in urine that contains albumin, but may be discovered when the urine is not albuminous. Different varieties of casts indicate particular kidney lesions. As previously mentioned, casts may be formed as a result of coalescence or degeneration of the cells or to the exudation of these cells or, perhaps, as a result of the infiltration of materials into the tubules. We therefore divide them into three groups: (1) Those formed by coalescence of cells; (2) those due to degeneration; (3) those due to some substance infiltrated into the lumen of the tubules either by an exudation from the epithelial cells or an infiltration from the blood.

Casts Due to Coalescence.—(*a*) Those formed by groups of epithelial cells, called epithelial casts; (*b*) those formed by red blood-corpuscles, called blood-casts; (*c*) those formed by leukocytes, called pus-casts; (*d*) those formed by bacteria, called bacterial casts.

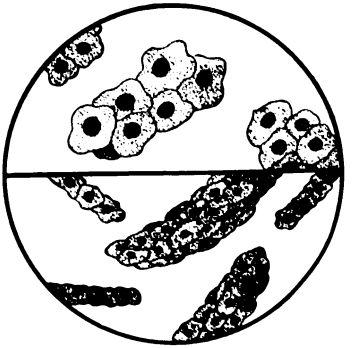
Casts Due to Degeneration of Cells.—(*a*) Those due to granular degeneration, called granular casts; (*b*) those due to fatty degeneration, called fatty casts; (*c*) those due to waxy disease, called waxy casts. (It is doubted whether these are due to degeneration or to infiltration of some substance into the tubule without marked change of the epithelial cells.)

Casts Due to Infiltration.—(*a*) Those probably due to an excretion of a hyaline material, called hyaline casts; (*b*) long ribbon-like cylinders, probably produced in a similar manner to the hyaline casts, called cylindroids; (*c*) unorganized casts, produced by the infiltration of crystalline or amorphous substances into the tubule, the most common of these being casts of urates.

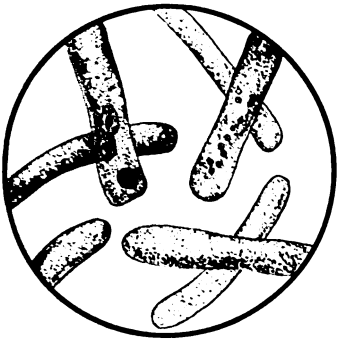
Epithelial casts are always significant of a kidney lesion. Blood-casts are found in acute parenchymatous inflammation or conditions in which the renal tubules become filled with red blood-corpuscles. Pus-casts also originate in conditions similar to the formation of blood-casts. Bacterial casts, particularly those composed of micrococci, are indicative of acute inflammation of the kidney, such as pyelonephritis. They may resemble granular casts, but differ from them in that they resist strong alkalies or acids. Granular casts may be composed either of coarse or fine particles. They are pale-gray or reddish-yellow in color, with irregular or wavy margins, the ends rounded and usually concave. Leukocytes, epithelial cells, and red blood-cells may be found adherent to these casts; they are indicative of nephritis. Fatty casts signify subacute or chronic lesions of the kidney; they result from fatty degeneration of the epithelial cells lining the tubules. Granular casts are frequently found to contain fat globules, and needles of fatty acids may also beset these casts. Under the microscope waxy casts appear as refractive, homogeneous cylinders; they are usually larger than other forms of casts; epithelium and red and white corpuscles may be found adherent to the surface. They are found in acute and chronic forms of nephritis, and in amyloid disease. They may give the reaction for amyloid material, but this is by no means constant. Hyaline casts as well as cylindroids probably result from the exudation or secretion of a material from the epithelial cells lining the tubules. The hyaline casts often contain upon their surface epithelial cells, leukocytes, red blood-corpuscles, and sometimes urates, oxalate of lime crystals, and bacteria. These casts have been found in the urine of healthy individuals as well as in the urine of patients afflicted with various forms of renal disease; they are, therefore, of little diagnostic value unless associated with other urinary findings, or when coated with epithelial cells, granular material, fat, leukocytes, or red blood-corpuscles. Cylindroids appear in ribbon-like cylinders. They are not indicative of kidney

Plate II.

Blood casts (some composed of disintegrated red blood-cells).

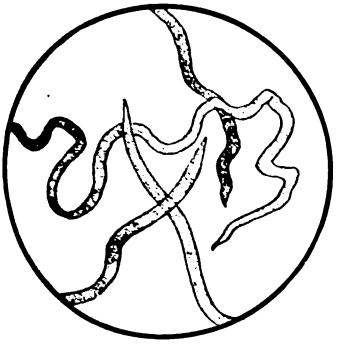


a. Squamous epithelium from the urine.
b. Epithelial casts.

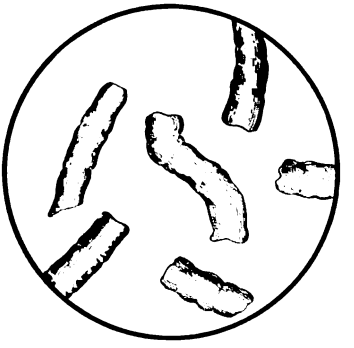


Hyaline casts.

Coarse and fine granular casts.



Cylindroids.



Waxy casts.

lesions, and are found in normal as well as abnormal urine. Unorganized casts are most commonly formed from urates. They are generally found in gout and renal congestion. They are of little significance.

Method of Examination for Casts.—The urinary sediment is obtained either by allowing it to stand for several hours or by the centrifuge. The sediment is placed upon a slide, and a cover-glass put upon it. Most forms of casts are easily recognized by microscopic examination. The hyaline casts are sometimes more readily demonstrated by staining with Lugol's solution.

Spermatozoa.—These are found in the urine of men after coitus, masturbation, and emissions. It must be remembered that they occur in the urine of women directly after coitus. This is often of medicolegal value, as in cases of suspected rape. They appear as elongated bodies, measuring about 50 microns in length; the head occupies about 4 or 5 microns, and is oval in shape; the remaining portion of the body has a tail-like elongation.

Parasites.—Occasionally echinococcus hooklets, distoma hæmatobium (the eggs of this parasite), and the embryo of the filaria sanguinis hominis may be present in the urine. The oxyuris vermicularis and trichomonas vaginalis may find their way into the urine from the vagina. Various forms of bacteria are occasionally found, such as the bacillus typhosus, the bacillus of tuberculosis, the streptococcus pyogenes, the diplococcus of gonorrhea, the pneumococcus, the bacillus of glanders, the ray fungus, and other micro-organisms. The appearance of tubercle bacilli in the urine signifies tuberculosis of some part of the genito-urinary tract. They may be confounded with the smegma bacillus, which is sometimes found in the urine, and to avoid error it is best to use the catheter in securing the urine. The urinary sediment that contains tubercle bacilli should be examined in the same manner as sputum that contains this organism. (For points of differentiation between the tubercle bacillus and the smegma bacillus, see the chapter on Bacteriology.)

Unorganized Sediments.

Uric Acid.—This occurs in the urine in crystals of a red or reddish-yellow color. They vary in size and frequently resemble whet-stone or sometimes rhombic plates, and are found in acid urine. They may be dissolved on the addition of

caustic soda or potash. This test is usually unnecess
their color is quite characteristic.

Urates of Soda and Lime.—Amorphous urates a
quently found in acid urine as a brick-dust sediment
color of this sediment may, however, be lighter wh
specific gravity is low. Under the microscope they ap
fine granules, sometimes in the form of casts. Th
particles can in no way be differentiated under the mic
from other fine granular matter, and it is therefore ne
to apply chemical tests for their determination. They
solved on the addition of acetic acid, or by the applic
heat. The *murexid test* may be employed for the d
of uric acid, a small amount of the sediment after d
placed upon a porcelain plate, and a few drops of ni
are added, which dissolves it; the solution is then
evaporated to dryness. A few drops of liquor of a
are then added, and when uric acid is present a purp
is developed.

Oxalate of Lime Crystals.—These crystals are
acid urine after partaking of certain fruits and vegetab
as apples, pears, tomatoes, beans, asparagus, etc., th
stances containing a large amount of oxalic acid. Th
tion is also met with in hypochondriasis, diabetes
catarrhal icterus, neurasthenia, and sometimes in other
diseases. They also occur in tuberculosis and can
health a few oxalate of lime crystals may be foun
urine, particularly after the ingestion of the foods m
above. When oxalates appear in the urine in large
the condition is known as *oxaluria*. The crystals o
of lime appear as transparent, refracting octahedra, c
bell crystals. They are soluble in hydrochloric a
insoluble in acetic acid.

Bilirubin and Hematoidin Crystals.—Bilirubin
may appear in acid urine as a crystalline or amorph
stance. In the crystalline form they appear either a
which are usually in clusters, or as rhombic plates.
of a yellow or red color. By the application of nit
green margin forms around them, and they are s
caustic potash. They have been found in the urine
affected by jaundice.

Hematoidin.—This substance closely resembles
When found in the urine it usually indicates some
hemorrhage in the urinary tract. Hematoidin ma

tinguished from bilirubin in that chemically it is insoluble in caustic potash, and on the addition of nitric acid a temporary blue color is developed.

Ammonia-Magnesium Phosphate, or Triple Phosphate.—This is found in weakly acid or more commonly in alkaline urine. These crystals appear as rhombic prisms or coffin-lid crystals. They are colorless and usually of large size. On the addition of acetic acid they are soluble. When they are found in the urine in large numbers the condition is known as *phosphaturia*.

Basic Phosphate of Magnesium.—These crystals may be found in feebly acid or alkaline urine, and appear as elongated rhombic plates, and are soluble in acetic acid.

Phosphate of Calcium.—When this substance is found in alkaline urine it appears as amorphous grains, which are soluble in acetic acid, but not by heat. When it appears in urine having a neutral reaction wedge-shaped prisms are found which are often massed in clusters. They are dissolved on the addition of acetic acid.

Sulphate of Calcium.—These are found in the urine as long needles or plates, being insoluble in ammonia and acids. They are of little clinical significance, and are rarely present in the urine. Amorphous deposits of this substance are sometimes encountered.

Cystin.—These crystals appear in acid urine as six-sided plates. They are insoluble in acetic acid, but quite soluble in ammonia. They are of clinical significance, since they occasionally give rise to calculi.

Leucin and Tyrosin.—These substances are usually found associated.

Leucin appears in acid urine in the form of small spheres.

Tyrosin.—This appears as fine needles which are grouped in bundles, sometimes called "Tyrosin sheaves." It is soluble in ammonia and hydrochloric acid, but insoluble in acetic acid. These substances are never found in normal urine. They are said to be due to degenerative changes of albuminoid products. They occur in acute yellow atrophy of the liver, phosphorus poisoning, and some of the infectious diseases, particularly enteric fever and variola. They have also been noted in leukemia and pernicious anemia.

Urate of Ammonia.—This substance appears in urine in the form of brownish balls, over the surface of which fine needles project, and they are commonly known as "hedge-

hog crystals." They appear in urine having an alkaline r tion, and are dissolved in hydrochloric and acetic acids.

Cholesterin.—Crystals of cholesterin appear in alka urine as plates or scaly particles. They are very rarely fou in the urine, but have been met with in cystitis.

Urea.—Urea is increased in fevers, also after the crisis pneumonia, after the ingestion of large amounts of albumino food, also in diabetes, and after exertion. It is diminished nephritis and in acute yellow atrophy of the liver, in wasti diseases and cachexia, especially when accompanied by drops and usually in starvation.

Test.—Fowler's hypochlorite method is based upon th fact that after decomposition of urine by hypochlorites decrease in the specific gravity will be noted. Every degre lost in specific gravity corresponds to 0.77 of 1%. Th process is as follows: Mix one volume of urine and seve volumes of hypochlorite solution (Labarraque's solution o chlorinated soda, U. S. P.). Before mixing this solution ascer tain the specific gravity of the one volume of urine. Find the specific gravity of the Labarraque's solution and multiply the result by 7. Add the specific gravity of Labarraque's solution and the specific gravity of the urine and divide by 8, which is the specific gravity of the mixture. After two hours, decomposition of the urine is complete, and the specific gravity of the mixture is now ascertained. Determine the difference in specific gravity before and after decomposition and multiply by 0.77, and the result is the percentage of urea.

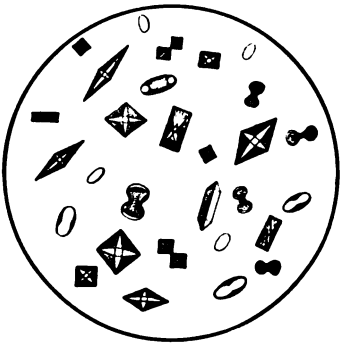
Chlorid of Sodium.—The quantity of chlorids is increased during the resorption of exudates and transudates, in intermittent fever (from the destruction of red corpuscles). The quantity is decreased in febrile diseases, particularly in croupous pneumonia during the stage of consolidation of the lung, also in nephritis and wasting diseases.

EXAMINATION OF THE FECES.

In an examination of the feces the frequency with which the stools are passed and the accompanying symptoms must be noted. The stools must be examined as to quantity, consistency, form, color, and odor, and as to their macroscopic and microscopic appearance.

PLATE III.

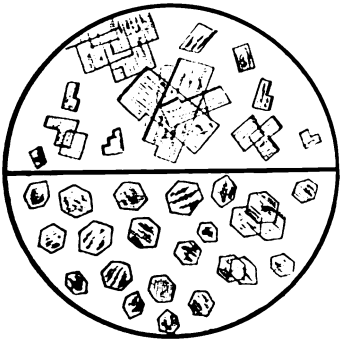
Uric acid crystals.



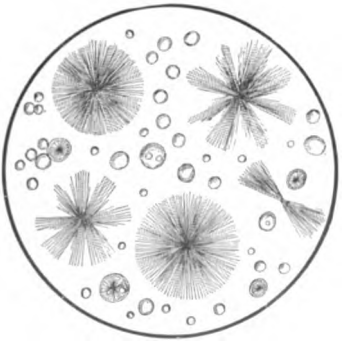
Calcium oxalate crystals.

Triple phosphate crystals.

Ammonium urate crystals.



a. Cholesterin crystals. *b*. Cystin crystals.



Leucin and tyrosin crystals.

Under normal circumstances the frequency of the intestinal discharges varies greatly in individual cases. Nursing children have from three to four movements a day, and under normal circumstances an adult has from one to two movements in the course of twenty-four hours, although two or three movements is not incompatible with health if the person experience no inconvenience. Constipation or, as it is called pathologically, *obstipation*, is the condition opposite to looseness or diarrhea. Under ordinary conditions it may be stated that the frequency of the discharges is particularly connected with the quantity of food taken; therefore, a person who is fasting will be constipated. Even the grade of the food has an effect upon the frequency of the intestinal discharges, but if the food pass rapidly through the intestinal canal, diarrhea may be produced.

Diarrhea.—Diarrhea is a most important symptom of intestinal catarrh. It may be due to cold and exposure, or it may result from infection, as in the acute infectious diseases (enteric fever, dysentery), or it may be due to errors of diet. Some drugs and poisons produce diarrhea, such as arsenic, mercury, etc. In cases in which the fluidity of the intestinal contents is increased diarrhea is apt to be an important symptom.

Constipation.—This is of diagnostic significance in peritonitis and in severe obstruction, as in stenosis of the intestine, fecal accumulations, intussusception, strangulation and invagination of the intestine, from tumors compressing the intestine, from constrictions, scars, or new formations in the intestinal wall, or from peritoneal exudations. In some conditions constipation may alternate with diarrhea, as in chronic peritonitis. Pain is present with the stools in inflammatory conditions of the intestine. Usually, severe pain occurs in inflammation of the lower portions of the rectum, in fissure, and abscess near the anus. Syphilitic disease and malignant affection of the rectum are always characterized by severe pain. Occasionally, severe pain is due to the presence of hemorrhoids. Painful straining at stool is known as *tenesmus*. Involuntary evacuations occur in any condition in which the cerebral functions are interfered with; they may occur in paralysis, and particularly in diseases of the spinal cord.

Macroscopic Examination of the Feces.—Amount.—The amount of feces passed by the normal individual in twenty-four hours varies between 120 and 180 grams, 75% of which

is made up of fluid and 25% of solid substances. C
diet the normal quantity of feces is reduced in amou
increased by starchy and vegetable diets. The an
feces per day is greatly increased in some diseases ;
Asiatic cholera 5000 grams in twenty-four hours ha
noted.

Color.—The normal color of the feces is a yellowish
or brown, the color being produced by urobilin, hydr
bin, or stercobilin. The feces of infants show a light
color. Spinach and cabbage produce a greenish appe
Iron, bismuth, and manganate cause a greenish-black
Occasionally, after the use of the iodids in large an
bluish particles will be noted in the stools ; from abse
bile in the feces they are of a light-gray color. When
transudations of serous material enter into the bowel,
water" discharges are observed ; these are characteris
Asiatic cholera, and are occasionally encountered in cl
nostras. If blood be present in the stool, it will have a
dish-brown or black color ; and if it has been retaine
some time, it frequently becomes tarry in appearance.

Reaction.—The reaction of the feces is, in the majorit
cases, neutral or alkaline. Upon an absolutely vegetable
an acid reaction may be encountered, due to fatty, acetic,
butyric acids. Occasionally, in children (nurslings) the s
is slightly acid.

Consistence.—Normally, the stool may be firm or sligh
mushy, although the form rarely has an independent va
Band-like, flat scybala may indicate stenosis of the bowel.

Odor.—In infants a slightly sour odor is normal. In ga
grenous discharges cadaverous, foul odors are encountere
this also occurs in dysenteric and syphilitic ulceration of t
rectum.

Character of the Stools.—Mucous Stools.—When appr
ciable quantities of mucus are present in the evacuations,
shows a catarrhal condition of the mucous membrane of th
intestine. In acute intestinal catarrh an abundant admixtur
of mucus, with thin stools, occurs. The same condition is
likely to be present in catarrhal dysentery. Not every small
slimy particle should be regarded as mucus in the stool.

Fatty Stools.—This shows a slightly glistening and greasy
appearance, and if large quantities of fat be present, it may
even be whitish or clay-like. This sometimes occurs in dis-
ease of the pancreas and in some forms of diabetes mellitus.

Bloody Stools.—These vary in appearance. If the blood has been thrown out in large amounts and quickly passed, it is apt to be bright-red in color, whereas if retained a long time in the bowel, it is dark, clotted, and tarry. Blood may result from bleeding hemorrhoids. If the blood is intimately mixed with the feces, it is either from the stomach or from the small intestine.

Purulent Stools.—Pus may occur in the stools from the rupture of an abscess anywhere in the intestinal tract. It is often found in inflammatory disease of the rectum. Pus may be present from dysentery, and from catarrhal, syphilitic, or carcinomatous ulcers of the large intestine.

Gall-stones and Enteroliths.—Gall-stones are noted in the stools, having found their way through the common bile-duct into the duodenum. When gall-stones are searched for, the stool must be passed through a sieve. If it is formed or mushy, it must be broken up by pouring water upon it. The gall-stone is easily recognized by its shiny appearance, smooth surface, and irregular outline, being faceted. Occasionally, after the eating of fruits, particularly pears, small concretions may be passed in the stool, which may be mistaken for gall-stones; they do not, however, give the characteristic reaction for those substances.

Solid portions of the feces, or undigested food, or concretions are called *enteroliths*. These are most frequently about the size of a cherry-stone, but occasionally are very much larger.

Shreds of Tissue and Fibrinous Casts.—Sloughs of necrotic tissue resulting from ulceration are not infrequently found in the stools. Sometimes pieces of new formations, particularly from carcinoma, make their appearance in the feces. Fibrinous casts and shreds of mucus are encountered, especially in the course of dysentery. The shreds of fibrin may be mistaken for worms, but careful examination will reveal their identity.

The animal parasites infesting the intestinal canal, and which are sometimes discharged in the feces, are described in the chapter on Animal Parasites.

Microscopic Examination of the Feces.—A small amount of the feces is mixed with distilled water or a weak solution of sodium chlorid, the solid portions being carefully broken into small particles. A little of this mixture is placed upon a slide and a cover-glass laid upon it, or a few drops of the mix-

ture are placed upon a cover-glass and this gently hea
dry, fixed by passing through a flame, and then staine
the latter mode of procedure various forms of epit
leukocytes, red blood-cells, and bacteria are demonstrat
the former method crystalline substances and various c
be examined. Under the microscope various forms
digested material, such as fat, starch granules, muscl
and other substances, can be investigated. For exam
of the ameba coli it is necessary to procure the fresh
tions, the examination being conducted upon a warm st

Red Blood-cells.—These are detected upon micro
examination. It sometimes happens in feces deeply s
with blood that it is difficult, or impossible, to find red b
cells, but in such conditions crystals of hematoidin are f

Leukocytes.—In suppurative conditions of the alime
canal, a great number of leukocytes are encountered i
feces. This is particularly so when ulceration exists. I
normal feces leukocytes are rarely if ever seen.

Epithelium.—In the feces during health epithelium is
stantly present, but when large numbers of these cells app
it is indicative of an intestinal lesion, particularly catarrhal
flammation. Various forms of epithelial cells are met w
and they often show degenerative changes.

Bacteria in the Feces.—Bacteria are always found in
feces in great numbers. Many varieties have been isolat
the most common being the bacillus coli communis, the bacil
typhosus, the spirillum of Asiatic cholera, the bacillus
tuberculosis, the spirillum of Finkler and Prior, the staphyl
coccus, the streptococcus, and others.

The following *crystals* have been found in the feces: Am
moniomagnesium phosphate, Charcot crystals, fat crystal
oxalate of lime, sulphate of calcium, and others.

PART I.

INFECTIOUS DISEASES.

THE CONTINUED FEVERS.

SIMPLE CONTINUED FEVER.

Definition.—Simple continued fever is a fever of two or three days' duration, dependent upon many causes. It is rarely fatal in temperate climates and is not contagious.

This is purely a symptomatic disease: it is not a substantive affection, and it is questioned by many pathologists whether such a disease actually exists. It is not an irritative fever, such as might occur from traumatism, nor is it due to the absorption of pus or other toxemias.

Synonyms.—Febricula; ephemera; synocha; ardent fever of the tropics.

Etiology.—There is no specific cause. Extremes of temperature may be said to produce the disease, and it has been claimed that it results from the inhalation of sewer-gas.

Overeating, mental and bodily fatigue, excitement, and violent emotions are supposed to be causative factors. The disease is more common in children than in adults.

Symptoms.—The disease rarely begins with marked chill; however, there may be slight chilly sensations; lassitude may occur early; and the temperature may rise to 103° F.; sometimes in children the temperature may be higher than this.

The face is flushed (herpes facialis may occur); the pulse is rapid, full, and bounding; headache, frequently pronounced, is present; often the tongue is coated or furred; and there is loss of appetite. The urine is scanty and highly colored, but

rarely contains albumin; the bowels are usually
but there may be diarrhea toward the close of
The patient may either be restless and suffer from
he may be dull and drowsy. The disease may ter
denly by crisis, or, more rarely, by lysis. The du
attack is from a few hours to several days. Wher
occurs in the tropics, the symptoms are more
temperature is much higher, the nervous sympton
pronounced, and the malady may be mistaken fo
Delirium and coma may occur, and the disease ma
fatally.

Diagnosis.—In the absence of inflammatory co
of traumatism, with the presence of the symptoms
erated, especially in children, a diagnosis of simpl
fever may be made.

Prognosis.—Invariably favorable, except in the
in the tropics.

Treatment.—This is purely symptomatic, very
cine being necessary. Usually, all that is requir
onset, a mild purge of calomel, in fractional dos
by a saline. Water should be given in moderat
forced feeding is unnecessary, as the disease is a n
of short duration. Should the temperature becon
the nervous symptoms prominent, cold sponging
may be beneficial. If the disease occurs in weal
tated persons, stimulants—preferably alcohol—sh
ministered.

INFLUENZA.

Definition.—A contagious fever due to a spe
the bacillus of Pfeiffer, usually occurring in epid
acterized by pronounced catarrh of the mucous
with marked debility, and showing a tendency to in
complications.

Synonyms.—Epidemic catarrhal fever; la grip

Etiology.—Predisposing Causes.—All races
are susceptible, the disease occurring in every clim
all seasons. Both sexes are affected, females, how
more predisposed. One attack increases the liabilit
It is highly infectious and contagious. Epidemics
last from eight to nine weeks. Sporadic cases a
occur for a long time after the active epidemic has

The **exciting cause** is the bacillus of Pfeiffer, which is found in the nasal secretions, sputum, and blood.

Incubation.—The period of incubation varies from a few hours to two or three days.

Pathology.—There is no characteristic lesion. Catarrhal inflammation of the mucous membrane of the respiratory and digestive tract and bronchopneumonia and lobar pneumonia are frequently met with. The specific organism has been found in the lung, liver, spleen, kidney, lymph-glands, membranes of brain, and endocardium, giving rise to inflammatory conditions. (For method of detection of the bacillus see p. 118.)

Symptoms.—Since the pandemic of 1889–1890, our knowledge of this disease has materially increased. The varieties and complexity of the symptomatology are so great that an accurate description of this disease is almost impossible. This has led to the classification and arrangement of different varieties, according to the prominence of certain symptoms. The disease begins after a very short period of incubation, generally with no or with ill-defined prodromes, with a pronounced chill followed by fever. Only in exceptional instances does the disease run its course without chill and fever. The range of the temperature is atypical, running from 99° F. to 105° F., rarely above this. The fever generally reaches its acme in the first twenty-four hours; the temperature may, however, rise stepwise, and reach its fastigium only after three or four days. The decline of the temperature also varies, the disease terminating by lysis or crisis; in the greater number of cases, however, the disease may be said to terminate by a form of rapid lysis. During convalescence the temperature may be subnormal. The duration of uncomplicated cases is about from five to seven days. In general terms, the poison of influenza attacks the mucous membrane of the respiratory and digestive tracts. It must, however, be remembered that there are cases that show no catarrhal phenomena; in fact, nothing but fever with rapid pulse, headache, and depression. The lungs may bear the brunt of the affection, although the digestive tract, the cerebrospinal system, or the heart may be the principal seat of the disease. This has led to the classification of four principal varieties: (1) The thoracic variety; (2) the cardiac variety; (3) the gastro-intestinal variety; (4) the nervous variety.

All these varieties show catarrhal symptoms in common, save in those exceptional cases already alluded to.

Then, following the chill and fever, there are pains in the limbs and back, sneezing, coughing (at first unproductive), injected conjunctivæ, pronounced headache, commonly referred to the root of the nose or behind the eyes, occasional vomiting, and diarrhea. Epistaxis is rare.

All these symptoms appear with extreme rapidity. Rashes occasionally occur, such as herpes, urticaria, or erythema. No matter how mild any of the preceding symptoms may be in an individual case, there are always great depression and weakness that are out of proportion to the intensity of all the other phenomena. This is characteristic of influenza. In the majority of cases early enlargement of the spleen may be observed. Cardiac asthenia is a prominent symptom, the pulse being rapid, weak, compressible, and sometimes intermittent. The urine is scanty, highly colored, and rarely contains albumin (toxic), generally without casts.

Complications.—The complications in this disease are extremely numerous. Respiratory complications are the most common: the so-called, and not well-understood, influenza pneumonias, bronchopneumonia, croupous pneumonia, and pleurisy, with and without effusion. Otitis media and complications relating to the heart, gastro-intestinal tract, and nervous system occur.

Sequels.—The more important sequels are those relating to the heart and lungs. Cardiac asthenia with an intermittent and irregular pulse has frequently been observed. Chronic bronchitis, emphysema, and tuberculosis result. Diabetes and neuralgia, especially of the fifth nerve, are frequent. Anemia, and even pernicious anemia, have been known to follow this affection.

Diagnosis.—The diagnosis depends upon the occurrence of an epidemic. The sudden onset, with chill and fever, the marked catarrhal phenomena, the peculiar headache, the intense depression, and the cessation of the symptoms in uncomplicated cases in from five to seven days by rapid lysis or crisis are diagnostic.

Prognosis.—In young, robust subjects almost invariably favorable. In the extremes of age, especially in the old suffering from chronic diseases, the prognosis is unfavorable.

Treatment.—Rest in bed, even in the mildest cases, is imperative. A laxative dose of calomel or a saline early is followed by good results, even in those cases in which diarrhea is present. For the pains, opium in some form is the most

reliable drug, and Dover's powder or minute doses of morphin are generally employed. The coal-tar products should not be administered on account of their depressing effects upon the heart. If given at all, they should be used cautiously and in very small doses. Diet is not important, as the disease is a brief one, but stimulation should be insisted upon from the onset, especially in old persons or in individuals suffering from some form of chronic ailment.

ENTERIC OR TYPHOID FEVER.

Definition.—An acute, specific, infectious disease of from twenty-one to twenty-eight days' duration, due to the bacillus typhosus; it is characterized by fever of a typical range, with gastro-intestinal symptoms, a rose-colored rash, and nervous symptoms. The disease shows constant pathologic lesions.

Synonyms.—Typhoid fever; gastric fever; nervous fever; infantile remittent fever; autumnal fever; typhus abdominalis.

Description.—This disease was first differentiated by Louis in 1829, before this time being usually confounded with typhus fever.

Etiology.—Predisposing Causes.—The geographic distribution is wide, the disease prevailing in every country; it is especially prevalent, however, in temperate climates. Autumn months particularly favor the development of enteric fever; often the disease follows hot and dry seasons. The majority of the cases occur between the ages of fifteen and thirty. The sexes are equally affected. Unfavorable hygienic surroundings, such as infected drinking-water, food, sewage, and poor drainage, predispose in susceptible individuals. Insects, especially the common house-fly, may be the carriers of the infective principle. One attack usually confers immunity.

Exciting Cause.—A bacillus discovered by Eberth, known as the bacillus typhosus. (For detection see p. 108.)

Pathology.—The cadaveric rigidity is well marked, the body is emaciated, and the typical eruption (rose-colored spots) is not present.

The bacillus, gaining entrance into the gastro-intestinal tract, finds its way through a rupture in the mucous membrane into the lymph-nodes of the submucosa, where inflammatory changes take place. The principal seat of the lesions is the lower part of the ileum, in Peyer's patches, but the solitary lymph-follicles of other parts of the intestinal tract may be

affected. Lesions have been found in the esophagus, stomach, vermiform appendix, and not infrequently in the large intestine.

For convenience of description the inflammatory changes may be divided into four stages, each lasting about a week: (1) The stage of infiltration; (2) the stage of necrosis; (3) the stage of ulceration; (4) the stage of healing.

The first stage, that of infiltration, may involve the whole or a part of the Peyer's patch. It is elevated, indurated, and of a gray color. Hyperemia may be noted around the infiltrated area. The surface of the infiltrated area is often irregular and contains darker areas, and is sometimes spoken of as the "shaven-beard" appearance.

Fig. 24.—Typhoid fever, showing necrosis of Peyer's patches and intense congestion of the bowel (modified from Kast and Rumpel).

The swelling of the Peyer's patch is due to the inflammatory exudate. The area contains numerous polynuclear leukocytes and round cells, many of which result from the proliferation of the fixed connective-tissue elements. There may be some red cells in the perivascular tissues, and the bacillus typhosus is also present. The blood-vessels show marked dilatation.

The infiltrated area rarely extends deeper than the muscular coat; in some instances, however, this is involved. The solitary lymph-follicles frequently reveal similar changes.

The second stage, that of necrosis, is due to the cutting off of the blood supply to the involved area, and the action of a specific poison. As the necrotic substance is discharged the

ulcer is formed, giving rise to the next stage. The necrotic process sometimes involves the walls of the blood-vessels, and when the necrosed area is discharged, hemorrhage is produced. The hemorrhage may be either open or concealed. If the ulceration be deep and the influence of the toxins upon the muscular coat of the bowel be pronounced, paralysis of the coats may take place; this is often the cause of concealed hemorrhage.

In the third stage, that of ulceration, the ulcer more or less conforms to the Peyer's patch. It is oval or circular in outline, the greater diameter being in the long axis of the intestine, opposite the mesenteric attachment. The floor is usually smooth, and formed by the muscular coat. Sometimes it is roughened, as necrotic tissue still adheres to the floor.

The edges may be somewhat overhanging and elevated. The great resistance of the muscularis mucosæ to the action of irritants gives rise to the overhanging character. The process may extend deeper, so that the muscular coat becomes necrotic; or in some cases the peritoneum may form the floor of the ulcer, and this may be involved, giving rise to perforation, which may be either circular or oval (punched out), the size varying from that of a small opening to two centimeters in diameter.

The perforation may be a slit-like opening, and results from the action of a peristaltic wave upon a scybalous mass lodging in the ulcerated area, the bowel becoming tense and the inelastic floor of the ulcer rupturing. When perforation occurs, the peritoneum invariably becomes inflamed, general peritonitis and death often following. If adhesions of neighboring coils of intestines are formed, a localized process results: frequently an abscess.

The fourth stage, that of healing or cicatrization, follows the stage of ulceration. Granulation tissue is formed, and finally the fully developed fibrous tissue, over which the epithelium ultimately spreads. The scar is smooth. It may be somewhat depressed, but does not show marked tendency of contraction; therefore, strictures of the bowel are almost unheard of in this disease.

The mesenteric glands are enlarged in all cases of enteric fever. They are soft and friable, and rarely ulcerate. The spleen is enlarged in about 90% of the cases. The capsule is tense, and the splenic pulp is soft and friable; in rare instances rupture has been found.

Granular degeneration of the voluntary and involuntary muscles of the internal organs, especially of the heart, is present. Hyaline degeneration of blood-vessels has been noted. Catarrhal inflammation of the gastro-intestinal mucous membrane occurs.

The specific organism has been found in the feces (rarely before from the tenth to the sixteenth day), in the urine, in the spleen, and in other internal organs.

The constitutional manifestations are due largely to the typhotoxins.

Period of Incubation.—This is from two to three weeks.

Symptoms.—The onset of the disease is insidious, and is preceded by prodromes, these consisting of malaise, vague pains in the limbs and back, headache, epistaxis, and slight evening fever. These symptoms continue until the patient is compelled to remain in bed. Diarrhea may be present, or may be easily invoked by a mild laxative. It is convenient to divide the symptomatology into periods of weeks, corresponding to the pathologic changes.

First Week.—At the end of the period of prodromes, which may be variable, the disease may be ushered in by chilliness or, rarely, by a distinct rigor. The pupils are dilated, appetite is lost, and the tongue is covered by a dry, white fur, and its edges and tip are red. Diarrhea continues; headache is increased, especially at night; the pulse is frequent, and its volume is good, but later it becomes dicrotic. The temperature is characterized by a gradual rise, being higher each evening by about a degree and a half, until the fifth or seventh day, when the fastigium is reached.

Toward the end of the week some tympanites occurs, and at this time a few scattered rales may be heard posteriorly over the chest. There is usually pallor of the face, with flushing of cheeks. The urine shows the changes of febrile conditions. At the end of the first week the spleen is perhaps slightly enlarged, and the characteristic eruption may be noticed.

Second Week.—The symptoms just described now become aggravated, with the exception of headache, which commonly disappears. The eruption, if not previously noticeable, now shows itself, perhaps on the abdomen, chest, or back, but rarely appears upon the extremities, and exceptionally upon the face. It consists of slightly elevated, rose-colored spots, from one to four millimeters in diameter, disappearing on

pressure, and reappearing when the pressure is relaxed. They appear in successive crops, which last from two to three days. The spleen is now found to be enlarged; the fever high and subcontinuous in type; the pulse weaker, from 90 to 120, and dicrotic. Occasionally, the hearing is dull. There may be low, muttering delirium. The intestinal symptoms are more pronounced than during the first week. In favorable cases defervescence may set in.

Third Week.—The symptoms become more severe; asthenia and emaciation are pronounced; fresh crops of the eruption may appear. At this time, which corresponds to the stage of ulceration, such complications as hemorrhage and perforation may be noticed. The temperature-curve becomes remittent in type; the pulse is feeble, and the first sound of the heart may be inaudible. Among the symptoms are excessive sweating and sudamina; dry and coated tongue, with brownish fur upon it; collection of sordes upon the teeth; and probably involuntary evacuation of urine and feces. The delirium now becomes more marked and perhaps violent in character, or there may be stupor, coma, carphology, or subsultus tendinum.

Fourth Week.—The symptoms ameliorate, the temperature becoming intermittent and the sordes disappearing as the tongue clears and the spleen contracts to its normal size. The urine increases in amount, and if there has been presence of albumin, this disappears. The mind clears, and great hunger develops. Convalescence is protracted, but may be interrupted by complications, relapses, and sequels.

The temperature during convalescence is very unstable, running a subnormal course; recrudescences may occur through constipation, excitement, improper food, etc.

Special Symptoms.—Temperature.—The temperature rises, gradually reaching its fastigium in from five to seven days. During the second week the course of the temperature is subcontinuous, falling each morning a degree or a degree and a half, and rising each evening to the same height as on the previous evening. This continues another week. During the third week there are greater remissions in the morning, the temperature assuming a decided remittent type until the fourth week, when it falls to or below the normal, giving an intermittent type. During convalescence the temperature is frequently subnormal, being labile and easily disturbed. Indiscretions in diet, visits of friends, excitement, mental emotions,

and constipation frequently produce a rise, called a recrudescence.

Departure from the type may occur. There may be a sudden rise at the onset, beginning with a chill, running a brief course, and ending by crisis. This is known as the abortive form, and takes place particularly in the enteric fever of children, showing a decided remittent range, thus giving rise to one of the synonyms called "infantile remittent fever." It is common for the typical curve to be interrupted by intercurrent diseases or complications. Hyperpyrexia, called the pre-

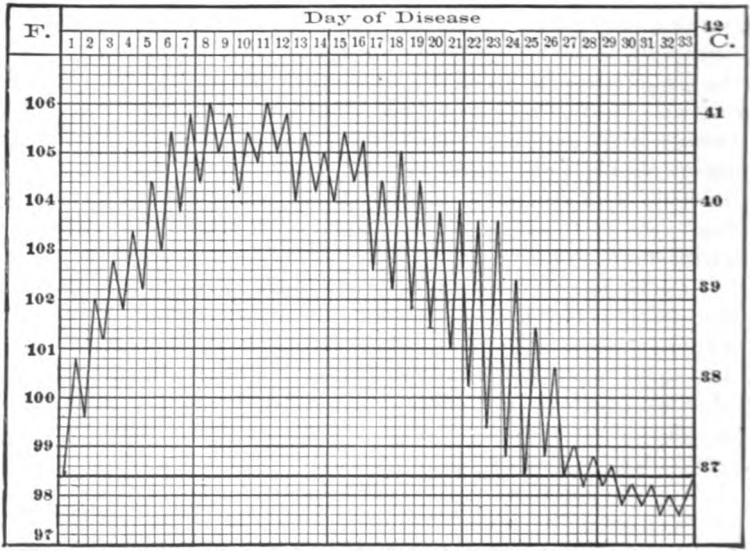


Fig. 25.—Temperature-curve in enteric fever.

agonistic rise, occasionally occurs, especially in fatal cases, just before death. The fever-curve of relapse corresponds to the original attack, but the fastigium is more quickly reached, defervescence taking place sooner.

Circulatory System.—As is usual in febrile diseases, the pulse frequency corresponds to the intensity of the fever, although this disease is one of relatively slow pulse. At the onset the pulse is of full volume and the tension is low, and soon in the process of the disease becomes dicrotic; this is an important diagnostic phenomenon in enteric fever. The pulse

frequency in uncomplicated cases is from 90 to 100, but in severe cases it may be accelerated. A pulse-rate above 110 is of unfavorable prognostic omen. There are changes in the heart muscle corresponding with those of the pulse. In severe cases the first sound of the heart becomes feeble or may be inaudible. Endocarditis and pericarditis, as complications, are of rare occurrence. Venous thrombosis, especially of the crural vein, often occurs during convalescence.

Blood.—Leukocytosis is absent in this disease, but any inflammatory complications, particularly peritonitis, gives rise to it. As the fever progresses anemia becomes more pronounced, being of a chlorotic type; this continues into convalescence and gives rise to "post-typhoid anemia." The Widal reaction is present in about 95% of the cases. (See p. 109.)

Respiratory System.—Epistaxis is an early and a common symptom; some degree of bronchitis is commonly met with, and in severe cases, from continuity of structure, the catarrhal process may extend downward, giving rise to bronchopneumonia. When coma occurs, food that is retained in the mouth and not introduced into its proper channel may get into the trachea, set up inflammatory changes, and give rise to a form of pneumonia known as deglutition inhalation, or insufflation pneumonia. This complication is one of extreme gravity. Croupous pneumonia also occurs as a complication. Pleurisy and empyema are occasionally met with.

Digestive System.—Nausea and vomiting may take place at the onset and continue for a few days. Epigastric pain, chiefly referred to the right iliac fossa, frequently occurs. Diarrhea is a characteristic symptom, and may be present from the first and continue throughout the disease, even into convalescence. Constipation is present in some cases, but diarrhea is easily invoked by mild laxatives. Stools may vary from two or three to a dozen or more in twenty-four hours. They are thin and brownish in color at first, but soon become yellowish, and are known as "pea-soup" stools. The tongue is characteristic: at first there is a slight whitish coat, appearing posteriorly, while the edges and tip are red; later the tongue becomes dry and the coating yellow and brown; at this stage it may also show fissures. Tympanites is common. Appendicitis may be a complication.

Hemorrhage.—Hemorrhage from the bowel is an important complication, and is found in about 5% of the cases, occurring late in the second or early in the third week, and varying from

a few drops to a quart or more. This gives rise to characteristic symptoms. Sudden fall in temperature; a rapid, running pulse; disappearance of nervous symptoms, such as delirium and coma; increased respirations, commonly called "air-hunger"; and perhaps the appearance of blood in the stool, are the diagnostic criteria by which this complication is recognized.

Concealed Hemorrhage.—If the blood does not appear in the stool with these symptoms, it is termed "concealed hemorrhage," and may be recognized by a sausage-shaped tumor in the right iliac fossa, which is dull or flat on percussion.

Perforation.—With a sudden fall in the temperature, reaching the normal or subnormal point, accompanied by abdominal pain, rigidity of the abdominal muscles, a rapidly running pulse, and hurried respiration, a diagnosis of perforation should be made. This is present in from 2 to 3% of the cases.

General Peritonitis.—Frequently, following perforation, vomiting sets in; abdominal pain becomes more intense and general, and leukocytosis is usually present. The abdomen is distended, the muscles are rigid and board-like, and the pulse is frequent and wiry. The temperature rises, flatus does not escape, and the lower border of liver dullness is obliterated; these symptoms indicate general peritonitis. Nervous symptoms are not marked.

Liver and Ducts.—Recent investigation has shown that the bile is often infected with the bacillus typhosus, and thus may be the cause of relapse. Jaundice is rare and very fatal.

Spleen.—The spleen is enlarged in 90% of the cases, and reaches its maximum size some time in the course of the second week.

Nervous Symptoms.—Headache is one of the early prodromes; it is usually frontal, but may be general; worse toward night, and subsides early in the second week. Somnolence, drowsiness, apathy, delirium, dizziness, dullness of hearing and vision, twitching of the tendons, and in grave cases stupor, which passes into coma, take place. Dilatation of the pupils may also be referred to the nervous system. Convulsions are rare, and only occur when meningitis or other complications take place.

Skin.—The rose spots constitute the specific eruption. They occur as scattered, pale-red, slightly elevated papules,

oval or irregularly circular in shape, and from one to four millimeters in diameter. They disappear on pressure and appear in successive crops, each individual crop having a duration of from two to three days.

They appear upon the abdomen, the upper and lower portions of the chest, the thighs, the shoulder-blades, and, exceptionally, upon the face. They are not very numerous as a rule, but may be abundant. A copious rash indicates a severe infection. The rash commonly appears at the end of the first or early in the second week, and continues throughout the disease, even into defervescence. Sudamina occur late: when sweating begins. Petechiæ are rare and of grave import. Herpes of the nose and lips takes place in some cases. A diffuse erythematous rash occasionally appears upon the face. Abscesses are complications. Furunculosis occurs in convalescence. Bed-sores appear in certain cases. The hair falls out during convalescence. Occasionally a bluish rash may appear upon the abdomen, and does not disappear upon pressure. This is known as the "tache bleuatre," and indicates body-lice. Purulent inflammation of the middle ear is a common sequel. The "tache cerebral" is present in some cases.

Urinary Apparatus.—The urine presents changes that accompany febrile diseases. The diazo-reaction is of some diagnostic importance. (See p. 159.) The toxicity of the urine is increased; especially marked after systematic bathing.

Relapse.—Relapses occur in from 3% to 18% of the cases, and at any time during the course of the disease, most generally during the period of defervescence. They are characterized by a return of all the symptoms; the course, however, is usually briefer than the original attack. Two, three, and even four relapses have been observed in a given case.

Varieties.—1. **Abortive.** The abortive form is of short duration, beginning abruptly by chill and ending by crisis. 2. **Mild.** The symptoms are slight, the temperature does not reach 103° F., the diarrhea is mild, the prostration is not great, and convalescence is rapid. 3. The **ambulatory form**—"*walking typhoid*" or "*latent typhoid.*" Symptoms so mild or resistance so great that the patient does not take to bed. These cases often terminate fatally. 4. The **apyrexial form.** This form runs its course without fever. 5. **Grave form.** Symptoms severe and usually associated with subcutaneous or internal hemorrhages; sometimes called the **hemorrhagic**

form. 6. **Infantile remittent fever.** Enteric fever in children is a common occurrence, and is characterized by fever of a remittent type, is often abortive, and the characteristic eruption is often absent. 7. **Enteric fever of the aged.** This disease is rare after forty years of age, but old people are occasionally affected. The mortality is high.

Intercurrent Diseases.—Cerebrospinal fever, malaria, tuberculosis, and syphilis are the principal intercurrent affections.

Sequels.—These are extremely numerous. Otitis media is common. Alopecia occurs, but the hair in the majority of cases returns. Phlebitis, especially of the left leg, appears. It may take place in both legs. A form of insanity, mostly due to anemia of the brain, may develop, from which the patient recovers when the general nutrition improves. Diseases of the bones are not at all uncommon, and even abscesses may take place in which the Eberth bacillus may be found months or even years after the attack. Dislocations and fractures of the long bones also occur.

Diagnosis.—This depends upon the occurrence of a prolonged course of fever, with abdominal symptoms, enlargement of the spleen, rose spots, headache, diarrhea, nosebleed, dicrotic pulse, and some rales in the chest. If constipation is present, diarrhea may be easily invoked by a mild laxative. The temperature may range from 103° F. to 105° F. The Widal test is of value in diagnosis, especially in doubtful or obscure cases. Ehrlich's diazo-reaction is present in enteric fever, but also takes place in other affections.

Differential Diagnosis.—The diseases mostly resembling enteric fever at some stage in its course are influenza, estivo-autumnal malarial fever, acute miliary tuberculosis, cerebrospinal fever, and ulcerative endocarditis.

1. Uncomplicated *influenza* lasts from five to seven days, whereas enteric fever shows its most prominent symptoms only at the end of the first week.

2. *Estivo-autumnal malarial fever* often closely resembles enteric fever. By the examination of the blood malarial parasites differentiate these diseases. (See article on Malaria.)

3. In *acute miliary tuberculosis* there is a history of previous cough or pleurisy. Temperature is more irregular, pulse is not dicrotic, rose spots are absent, pulmonary symptoms are more marked than abdominal, and examination of sputum, stools, and urine may show presence of tubercle bacilli.

4. *Cerebrospinal fever* may resemble enteric fever at its onset, but the absence of typical temperature, rose spots, and the greater preponderance of nervous symptoms, such as headache, retraction of the muscles of the back of the neck, and cerebral vomiting will usually disclose the true nature of the affection.

5. In *ulcerative endocarditis* the previous history, presence of changeable endocardial murmurs, chill, fever, sweating, and leukocytosis are important differential points.

Prognosis.—This depends in a great measure on treatment. The systematic cold-bath treatment of Brand has reduced the mortality to about 7%. The earlier the treatment is instituted, the more favorable is the prognosis. The occurrence of severe complications is unfavorable. Fat subjects bear the disease badly.

Treatment.—Prophylaxis. It is important to prevent new cases from developing, either from direct or indirect contact. Absolute destroying of the discharges and disinfection of the soiled linen must be carefully attended to. Chlorinated lime, commercial hydrochloric acid, and solutions of carbolic acid may be employed for disinfecting. Corrosive sublimate is less efficient, as it coagulates albuminous matter.

Rest.—Rest in bed.

Diet.—*There should be an absolute liquid diet*, milk, broths, etc. Fluids should be administered in liberal amounts; alcohol forms no necessary routine in the treatment of this disease. It should be given in cases of great prostration and on the appearance of grave nervous symptoms.

Routine Treatment.—A laxative dose of calomel should be given before the tenth day, *never later*. Specific treatment is of no avail.

Method of Brand.—Systematic cold bathing should be resorted to as early as possible. When the temperature in the axilla reaches 101⅗° F., a cold bath is given and repeated every three hours, the water being at the temperature of about 70° F. The patient should be immersed in the tub, the water covering all but the head. Then water of a lower temperature should be poured upon the patient's head, or a wet ice pack used. Gentle friction should be applied constantly by the attendants, and the patient should be encouraged to do likewise. *Do not rub the abdomen!* The duration of the bath is fifteen minutes. Some alcoholic stimulant should be administered to the patient before and after the bath. When the bath

is finished, he should be lifted back to his bed and covered with woolen blankets. The temperature should be taken one-half hour afterward. Contraindications to the bath are *hemorrhage* and *perforation.*

Intestinal Hemorrhage.—There should be absolute quiet and withholding of food for a time. Opium in some form should be given to its physiologic limit, ice-bags placed upon the abdomen, and the foot of the bed elevated. Hot-water bags may be applied to the extremities.

Peritonitis.—Peritonitis also calls for the full administration of opium and of ice-bags to the abdomen. In perforation surgical interference should be solicited if diagnosticated early, otherwise this condition must also be treated with liberal doses of opium.

Heart Failure.—Heart failure sometimes occurs in convalescence. The treatment is absolute quiet in the recumbent posture. Cardiac stimulants, such as alcohol and strychnin, should be freely administered.

Constipation.—This may be relieved by enema and glycerin suppositories. *Avoid purgatives.*

Tympanites.—Give alcohol freely. Apply turpentine stupes to the abdomen. Turpentine internally is of use. The bowel should not be perforated by hypodermic needle or other appliances.

Management of Convalescence.—The process is long and tedious. Semisolid or solid food should not be administered before the evening temperature has been normal for at least one week. If the *anemia* is pronounced, iron, arsenic, cod-liver oil, and quinin are of use.

TYPHUS FEVER.

Definition.—A specific, infectious, contagious disease, occurring in epidemics, usually of short duration,—fourteen days,—characterized by marked nervous symptoms, a typical rash, and a high mortality.

Synonyms.—True typhus; petechial typhus; ship fever; jail fever; spotted fever; typhus exanthematicus.

Etiology.—A disease of cold and temperate climates, occurring especially in the British Isles and in Southeastern Russia. This was the great fever of the past historic epoch. The disease is markedly contagious as well as infectious, although it is probable that the contagion is not carried to

any great distance. It spreads through the atmosphere, and is carried by fomites. Overcrowding, filth, and scarcity of food favor its development. The largest number of cases occur between the ages of fifteen and twenty-five, although it may appear in childhood and in old age. Both sexes are equally affected. The exciting cause is unknown; no doubt, however, it is a specific germ. Several organisms have been described, but none is generally accepted. One attack confers immunity.

Pathology.—There is no characteristic lesion. The post-mortem rigidity lasts but a short time. The typical eruption

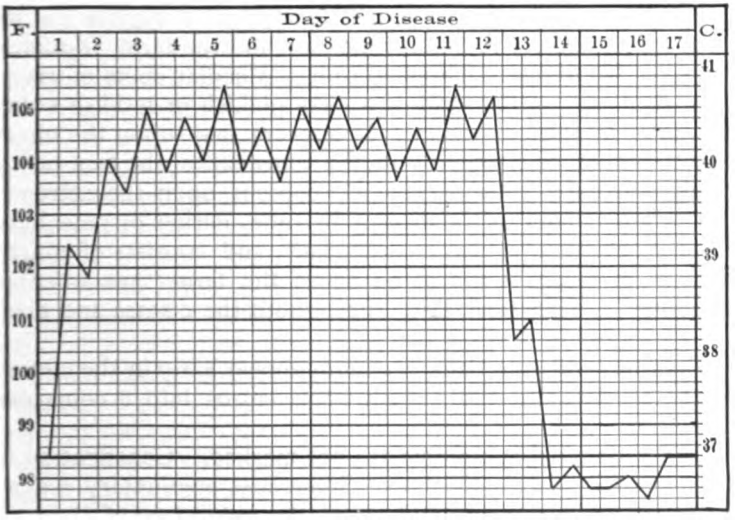


Fig. 26.—Temperature-curve in a case of typhus fever.

persists. Spleen and liver are found enlarged, and the blood is profoundly altered, dark, and stains the tissues. No ulcers are found in the intestinal tract.

Period of Incubation.—The period of incubation is a variable one, but is generally estimated as being about fourteen days.

Symptoms.—The prodromes are usually slight and often entirely absent, the disease beginning with a severe chill accompanied by nausea, vomiting, and epigastric pain. The temperature rises abruptly, and soon reaches 104° F. or 105°

F., or even higher. The pulse frequency is increased, being over 100 a minute, hard, and not easily compressible. The patient has the sensations of an impending severe illness, becomes weak, vertigo sets in, and soon delirium follows. There are deafness and tinnitus aurium. The face is reddened or turgid, the eyes are glassy, the pupils contracted, and the conjunctivæ injected. A mousy odor exudes from the body. The tongue is coated with a grayish-yellow fur; the lips are dry and cracked and bleed easily, and the teeth are covered with sordes. Anorexia is complete, but there is much thirst. The liver and spleen are painful upon palpation, and are now found to be enlarged. These symptoms continue for about five days, when the specific eruption appears.

Eruption.—The characteristic eruption of typhus in its early stages closely resembles measles, and the term "measley eruption" is frequently applied to it. Macular spots of irregular size and outline and of a dirty pinkish or reddish color characterize the exanthem. It appears first upon the chest and abdomen, and extends to the extremities, the face being rarely affected. It is particularly copious upon the extremities, where it later in the disease becomes darker or petechial. Another eruption is also characteristic, and consists of marbling or mottling of the skin; hence the term "subcuticular mottling." This rash lasts throughout the disease and does not disappear in death.

Delirium and subsultus tendinum appear early in the course of the disease, and the patient soon lapses into a comatose condition.

The abdominal symptoms are not marked. Constipation is characteristic of this affection. Sometimes involuntary evacuations take place, from paralysis of the sphincters of the rectum and bladder. The pulse is rapid,—120 to 140,—small, and soft. The respirations are frequent,—40 to 60,—noisy, and blowing in character. As these symptoms occur, changes take place in the eruption, in the center of which purplish or bluish points appear ("true petechia").

The patient may now lie in a semi-unconscious or comatose condition for some days. If recovery takes place on or about the fourteenth day of the disease, a sudden drop in the temperature occurs,—frequently five or six degrees,—with copious sweating, free diarrhea, or the passage of large quantities of urine. The temperature falls to normal or subnormal, the pulse becomes slower and fuller and less frequent,

nervous symptoms entirely disappear, and the patient rapidly passes into convalescence.

Urinary Symptoms.—The urine shows the usual characteristics of febrile urine. The quantity is lessened, the specific gravity high, and there is abundance of solid material, such as phosphates, urates, and toxic albuminuria.

Complications.—If this favorable issue does not take place, the patient remains in the comatose condition. Complications develop, particularly hypostatic congestion of the lungs, croupous pneumonia, pleurisy, pleurisy with effusion, and the patient may die from gangrene or from edema of the lungs. Relapses are extremely rare.

Sequels.—Peripheral neuritis, thrombosis of the veins, parotid bubo, and otitis media occur as sequels.

Diagnosis.—This is not difficult; the sudden onset with high temperature, the appearance of the rash on or about the fifth day, the marked nervous symptoms, the contracted pupils, the early prostration, the history of the epidemic, are all characteristics of this disease.

Differential Diagnosis.—The diseases which most closely resemble typhus fever are relapsing fever, measles, and enteric fever. The differential diagnosis between typhus and relapsing fever can readily be made by an examination of the blood. In relapsing fever, during the febrile stage the specific organism, the spirillum of Obermeier, is found. The disease can be differentiated from measles by the absence of catarrhal symptoms, the sparsity of eruption of the face, and the severer nervous symptoms. The differential diagnosis between typhus fever and enteric fever may be made from the greater predominance of intestinal symptoms in enteric fever, the typical temperature-curve, the eruption occurring later and disappearing upon pressure, and from the greater length of the disease. The Widal reaction is of great use in the differential diagnosis in obscure cases.

Prognosis.—Typhus fever is a very serious affection, the average mortality being between 30% and 40%. Unfavorable signs are a soft, compressible pulse above 120, hurried respirations, pinhole pupils, convulsions, muscular tremors, hiccup, and the presence of serious complications.

Treatment.—The treatment is symptomatic. Prophylaxis is of the greatest importance. An abundant supply of good food, fresh air, and free ventilation are important.

Stimulation is necessary on account of the severe prostra-

tion. If hyperpyrexia occurs, the cold bath should be resorted to. Strychnin may be necessary as a respiratory and cardiac stimulant. Complications must be treated as they arise. During convalescence the patient should be kept upon his back, and not allowed to assume an upright position too early, on account of the cardiac asthenia. Constipation should be relieved by enemata. Tonics should be administered.

RELAPSING FEVER.

Definition.—An acute, specific, infectious disease, markedly contagious, prevailing in epidemics and in times of famine. It is characterized by three stages: First, a febrile stage, lasting about seven days; second, a period of apyrexia; and third, a relapse on or about the fourteenth day, with the initial symptoms reappearing. This disease is due to the spirillum of Obermeier.

Synonyms.—Spirillum fever; famine fever; and typhus recurrens.

Etiology.—The disease is common in Ireland and in some parts of Europe. It is rare in this country. It occurs at all seasons of the year, and is more common in children and in early adult life, and is seldom found in persons after fifty. Sex and occupation are without influence. Famine is a predisposing cause.

Exciting Cause.—Obermeier, in 1873, observed in the blood of patients suffering from this disease highly motile spiral filaments. These organisms have since been constantly found in the blood of patients suffering from relapsing fever. They have a corkscrew shape, and move with great rapidity in a rotary manner. They may adhere to the blood-corpuscles, but are occasionally found clumped or in masses. They do not occur during the period of apyrexia in the peripheral blood, but reappear again in the relapse. These micro-organisms have not been found in the discharges, in the saliva, or in the sweat, nor can they be cultivated outside of the body. Inoculation experiments have been successful. The organism has not been found after death. (For description of the germ see p. 117.)

Pathology.—The parasite finds access to the body in some manner not yet understood, and multiplies, giving rise to the characteristic phenomena of the attack. One attack does not confer immunity.

Leukocytosis is common, but there are no constant anatomic lesions. Cadaveric rigidity is well marked, and the body is usually emaciated on account of the high temperature. The liver and spleen are enlarged. The heart is not changed. The lungs may show the appearance of bronchitis or bronchopneumonia.

Symptoms.—Incubation.—The period of incubation is from five to eight days. The onset is abrupt, with chill, frontal headache, pain in the back and limbs, and temperature rising to 104° F. The patient suffers from severe thirst and anorexia. The tongue is covered with a whitish fur, and the temperature

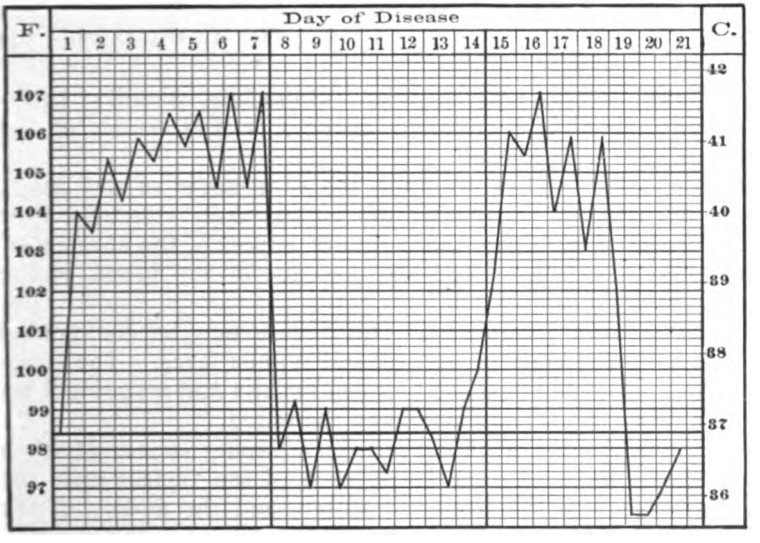


Fig. 27.—Temperature-curve in a typical case of relapsing fever.

may continue rising to 106° F., or even higher in the evening, with morning remission of a degree or two. Chilly sensations may continue, and sweating occurs. The respiration may be thirty a minute or even higher. Jaundice occurs in about 75% of the cases.

The urine may be stained with bile pigment; tenderness is present over the liver and spleen, and these organs are found to be enlarged. No typical eruption occurs. Sleeplessness is usual; the mind is clear, but delirium may ensue at the end of the attack. Crisis occurs about the seventh day, with pro-

fuse sweating, temperature and respiration falling, and the pulse-rate rapidly decreasing from 130 to about 70. The tongue clears, and the patient may feel comfortable. Recovery is rapid.

The second period, or period of apyrexia, lasts about seven days. The temperature, which may have been subnormal, rises in a day or two; ravenous hunger develops and strength is speedily regained, when, on or about the fourteenth day from the beginning of the disease, the temperature rises again with all the symptoms of the initial attack. The relapse differs from the original attack in the fact that the fever, although it may be higher, is usually briefer. A second, third, or even a fourth relapse has been observed. Convalescence is slow and tedious, and some time elapses before the patient regains his normal condition.

Complications and Sequels.—Bronchitis, pleurisy, bronchopneumonia and croupous pneumonia, and edema of the lungs have been noticed.

Diagnosis.—Depends upon the suddenness of the onset, the high febrile movement, the critical defervescence on or about the seventh day, the period of apyrexia, the absence of eruption, the relapse, and the presence in the blood of the specific organism.

Differential Diagnosis.—The disease may be confounded with irregular types of malaria and typhus fever. The diagnosis may readily be made between malaria and relapsing fever by a microscopic examination of the blood.

The differential diagnosis between relasping fever and typhus fever may be made from the absence of the typical rash, the enlargement of the liver and spleen, and the finding of the specific organism in the blood.

Prognosis.—This is favorable, the mortality being very low. Death ensues from complications.

Treatment.—The treatment is purely symptomatic. No drug is known that will abort or prevent the relapse. Antipyretics are usually not required. For the pain in the epigastrium and liver or spleen, cold or warm compresses and opium may be employed. Tonics should be given during convalescence.

YELLOW FEVER.

Definition.—An acute, specific, epidemic disease of short duration, occurring in tropic or subtropic countries. It is characterized by epigastric tenderness, albuminuria, the vomiting of black, altered blood, and by a yellow discoloration of the skin, due to the bacillus of Sanarelli.

Synonyms.—Black vomit. Known as yellow fever in all languages.

Etiology.—A disease of populous centers extending along the lines of travel. One attack usually confers immunity. All ages and both sexes are equally affected. The disease is endemic in the West Indies, parts of the Mediterranean coast, South America, and Africa. This is known as the *focal zone.* It is carried to the Gulf States and occasionally as far north as Virginia; this is known as the *perifocal zone.* When it invades cities further north, it is known as the *zone of accidental epidemics;* such places include New York, Philadelphia, and Boston. It is extremely rare above the fortieth parallel of latitude.

Exciting Cause.—The bacillus of Sanarelli. (See p. 120.)

Period of Incubation.—Varies greatly, it may be from one to seven days.

Pathology.—There is present marked jaundice. The liver shows marked fatty degeneration. The kidneys reveal parenchymatous or fatty degeneration. In the stomach a black fluid is found; the walls of this organ exhibit areas of hemorrhage, and microscopic examination will show some fatty degeneration. The heart is flabby, and may also reveal fatty degeneration.

Symptoms.—The disease usually consists of three well-defined stages: the period of *onset,* the *calm,* and the *collapse.* The disease begins with a chill or chilliness, followed by muscular pains, headache, and pains in the back. The temperature rises rapidly to its maximum in twenty-four hours. It may reach 103° F. or 104° F. The pulse is usually from 70 to 80; the face is flushed; the eyes are clear and glistening, with some edema of the lids; the expression is anxious. Nausea and vomiting are common. True albuminuria occurs early on the second day. Slight jaundice may be present at this time. There is pain in the epigastrium, and the bowels are constipated. It is characteristic of this disease that the pulse diminishes as the fever continues, so that bradycardia

may be a symptom of this disease. This stage lasts from three to five days, and is succeeded by the stage of calm. From this point on convalescence may be rapid and uninterrupted, or the patient may pass into the third stage. The calm rarely lasts longer than from twelve to twenty-four hours.

In the stage of collapse all the initial symptoms return with greater severity. The pulse now becomes more rapid and the fever higher, although this is not a disease characterized by hyperpyrexia. Jaundice, if not previously present, makes its appearance, from which the disease receives its name. The vomiting becomes copious, and hemorrhages occur. They may be from the nose, lungs, bowels, or, more commonly, from the stomach (the characteristic black vomit). The black vomit consists of altered blood, with parts of the gastric contents. Nervous symptoms now appear; the mind, which has remained clear up to this time, becomes clouded, and the patient dies in coma. Recovery may even follow some of the severe symptoms just enumerated, but this is most unusual. The duration of this stage is indefinite, lasting a week or more.

Types.—Mild, severe, or grave (hemorrhagic) cases occur during an epidemic.

Diagnosis.—This depends upon the knowledge of an epidemic, the disproportion of pulse to temperature, the early albuminuria, the yellow discoloration of the skin, and the black vomit. The agglutination test may be useful.

Differential Diagnosis.—This disease must be differentiated from dengue, in which the prominence of pain in the joints and bones coming on suddenly, with high fever and rapid pulse, are seen; absence of albuminuria; grave gastric symptoms, and hemorrhages. Jaundice is not present.

Prognosis.—Mortality is high, in some epidemics reaching 50%. Unfavorable symptoms are jaundice, hemorrhages, rapid pulse, and high fever.

Prophylaxis.—Isolation of all cases and even of suspected cases, as the disease is both contagious and infectious.

Treatment.—Disinfection of the person and effects. Strict quarantine. There is no known means of aborting the attack. Patient should be placed in bed, and have absolute rest, good ventilation, and hygienic surroundings. A mild cathartic should be given at the onset. Calomel is perhaps the best drug. Vomiting and gastric irritability may be treated by carbolic acid, cocain, or broken doses of calomel. Dry iced champagne is of use in some cases. The fever should be

treated by cold applications to the head and cold sponging. For the pain opium in some form is the most useful drug. Strychnin is of use in cardiac asthenia. Acidulated drinks in small quantities are grateful to the patient. For the hemorrhage opium, ergot, and suprarenal capsule extract may be used.

DENGUE.

Definition.—An acute, infectious, febrile disease of short duration, prevailing in epidemics in warm climates, characterized by an active febrile movement, with remissions, intense headache, pain in the joints and muscles, cutaneous eruptions, and low mortality.

Synonyms.—Breakbone fever; dandy fever; broken-wing fever.

Etiology.—This disease is markedly contagious, occurring in widely spread epidemics in tropic and subtropic countries, at all ages, in both sexes, and attacks the majority of the population. The specific cause has not been definitely determined. The disease appears particularly in seaport cities.

Period of Incubation.—From two to five days.

Symptoms.—The disease begins with marked chill and rigors, and the temperature rises abruptly. There is pronounced headache, particularly in the temporal regions; intense pain in the back; and characteristic pain in the joints and bones. This produces a marked stiffness and a corresponding gait; hence the synonyms breakbone fever and dandy fever. Gastric symptoms are marked; tongue is thickly coated; there are complete loss of appetite and marked thirst. In this stage eruptions may occur: they are usually of a vasomotor nature. The face is red, the eyelids are swollen, and the conjunctivæ are injected. The prostration is extreme. This stage lasts about three days ("three-day fever"). Occasionally, the temperature may fall at the end of the first or second day, either by lysis or by crisis, and when by the latter, there is profuse sweating. This is followed by a period of apyrexia, and eruptions are likely to show themselves: erythema, urticaria, herpes, etc. The fever rises again to its former height or even higher, but terminates rapidly, lasting about two or three days. Irregular cases and cases in all degrees of severity appear during an epidemic.

Diagnosis.—This depends upon the presence of an epidemic, abruptness of the onset, early occurrence of the joint

and muscle pains without redness and very little swelling, remissions during the course of the attack, with eruptions, swelling of the glands, and profound prostration.

Differential Diagnosis.—Differential diagnosis must be made between dengue, influenza, and yellow fever.

	Dengue.	*Influenza.*	*Yellow Fever.*
Where occurring	In tropic and subtropic countries.	In all countries.	In tropic and subtropic countries.
Numbers affected	Large majority of population.	Large majority of population.	Cases limited.
Nature	Contagious.	Contagious.	Contagious.
Duration of epidemic	Two to five months.	Six to eight weeks.	Indefinite.
Affects whom	All races.	All races.	Foreigners particularly.
Period of incubation	Two to five days.	From a few hours to a few days.	One to seven days.
Characteristics	Severe pains in joints and muscles.	General muscular pains.	General muscular pains.
Ambulatory cases	Frequent.	Frequent.	Rare.
Catarrhal symptoms	Extremely rare.	Characteristic.	No catarrhal symptoms.
Pneumonia or pleurisy	Rare.	Frequent.	Rare.
Gastric symptoms	Prominent.	Not so prominent.	Nausea and vomiting characteristic.
Diarrhea	Rare.	Frequent.	Constipation.
Spleen	Not enlarged.	Enlarged.	Slightly enlarged.
Pulse and temperature	Rapid and high fever.	Rapid and high fever.	Pulse slow and temperature not so high.
Eruptions	Frequent.	Rare.	Jaundice.
Hemorrhages	Rare.	Rare.	Common.
Albuminuria	None.	None.	Constant.
Mortality	Low.	Somewhat higher.	Very high.
Sequels	Rare.	Common, frequent.	None.

Rheumatic fever may resemble dengue, the acid sweats, cardiac complications, the less severe onset, the symmetric involvement of the joints, with redness and swelling, all pointing to acute rheumatic fever.

Prognosis.—The disease is rarely fatal.

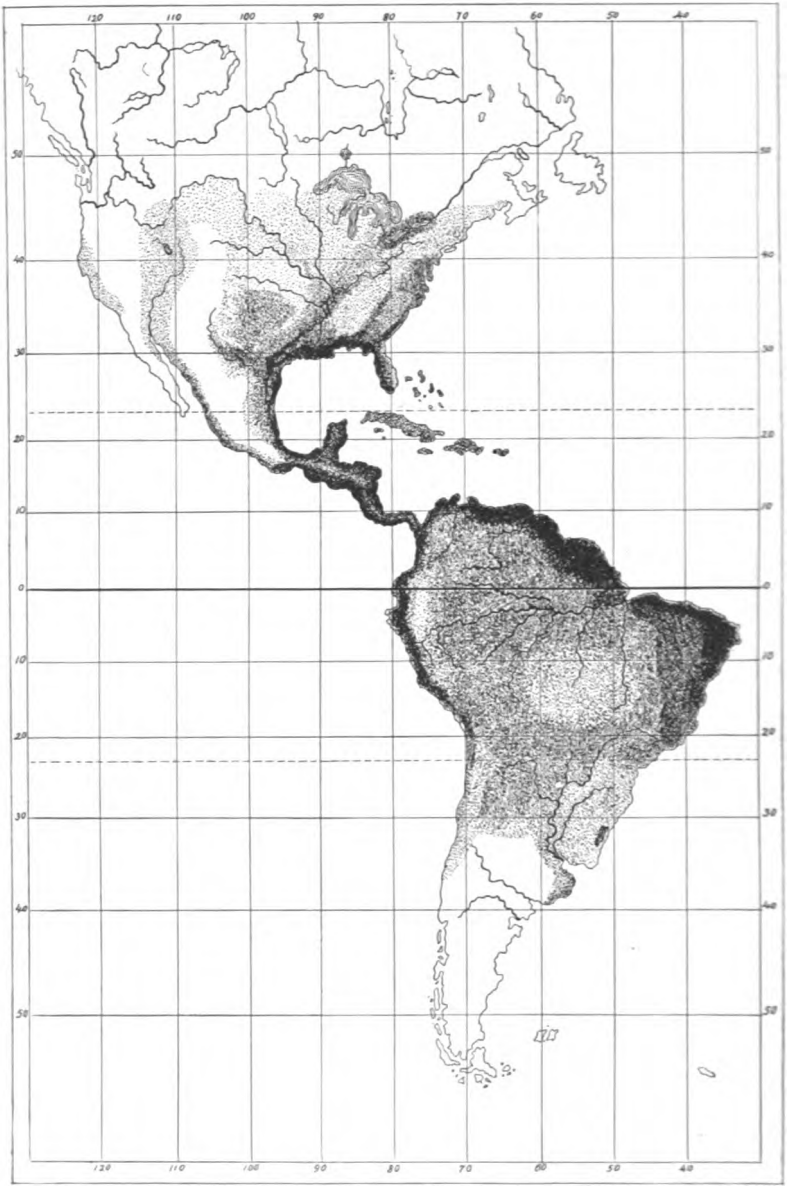
Treatment.—No specific treatment. A purge should be given at the onset and opium and its derivatives to relieve pain. Antipyretics may be of use. Administration of quinin is without avail. During convalescence the patient should have an abundant supply of nutritious food and tonics.

PERIODIC FEVERS.

MALARIA.

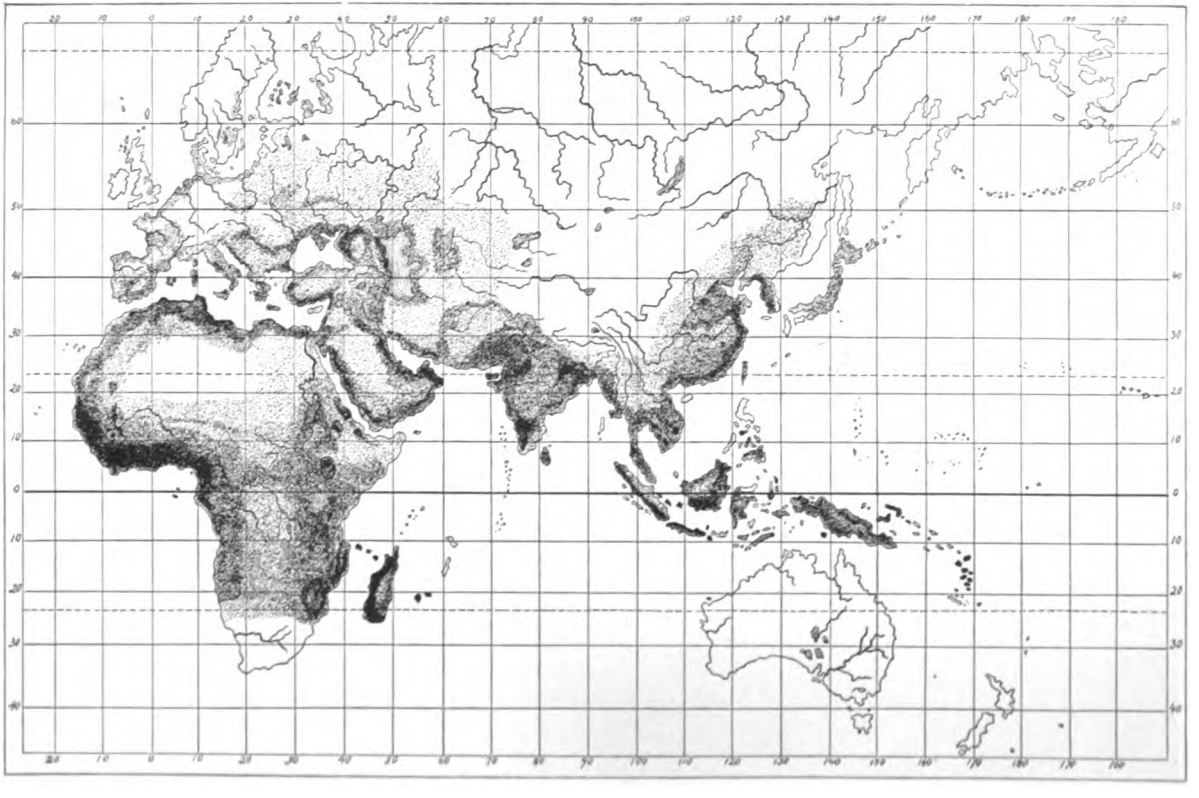
Definition.—Malaria is an infectious disease, characterized by periodicity of symptoms; anatomically, by enlargement of the spleen, and in the blood by the presence of the specific parasite.

Plate IV.



Showing the distribution of malaria as indicated by the shading (modified from Mannaberg, "Die Malaria Krankheiten").

PLATE IV.



Showing the distribution of malaria as indicated by the shading (modified from Mannaberg, "Die Malaria Krankheiten").

Groups.—Tertian fever, quartan fever, and estivo-autumnal fever. The tertian and quartan fevers are usually intermittent, and the estivo-autumnal fever is remittent or continued.

Synonyms.—Malaria literally means "bad air." Malaria; chills and fever; ague; intermittent fever; remittent fever; swamp fever; chagras fever; Panama fever; Roman fever; African fever; black-water fever.

History.—Meckel, in 1847, found in the blood of a cadaver dead of malarial fever oval and round pigmented bodies that were probably the specific parasite. In 1849 J. K. Mitchell, of Philadelphia, found pigment in the blood of a malarial patient. Lewis, an English observer, found hematozoa in the blood of rats. To Laveran, a French army surgeon, is due the credit of the discovery, in 1880, of the malarial parasite.

Etiology.—Predisposing Causes.— Malarial fevers are prevalent in most parts of the world. They are, however, mostly a disease of tropic and subtropic countries, and appear much less frequently in temperate zones.

Seasons.—In the tropics malarial fevers are endemic throughout the year. In subtropic and temperate climates they are most frequent in August, September, and October, then decreasing in severity toward December and increasing again in the early months of the year, until they reach their maximum about September. In the early months of the year the infections are rather mild, the tertian and quartan types being usually single and most frequent; the severer ones in the early fall, being usually double tertian, quartan, and estivo-autumnal.

Influence of Soil, Moisture, and Altitude.—Lowlands, river-bottoms, and swamps favor infection. Decaying vegetable matter also seems to predispose. In mountainous regions malaria is of rare occurrence. Cultivation of the soil and drainage seem to lessen the liability of contracting the disease. It is supposed that winds play some part in its spread.

Race, Occupation, Age, and Sex.—The white race is most susceptible; negroes and Indians to a lesser degree. Occupations that expose individuals to moisture and contact with the soil, such as laborers upon public works, road-making, gardening, fruit-gathering, etc., predispose. Most frequent in early adult life, less so in the extremes of age. Women are less frequently attacked, being less exposed. (It has been demonstrated by Thayer that congenital malaria probably does not exist.)

Exciting Cause.—Plasmodium malariæ, or hematozoon of Laveran. This parasite belongs to the group of protozoa. It has not been demonstrated in soil or water, and all attempts at cultivation have failed. It has, however, been found in the bodies of certain species of mosquitos. The mode of entrance into the blood of man has not been clearly demonstrated, except perhaps by infection from mosquitos. It is supposed to gain entrance into the human economy through the respiratory tract, the digestive tract, and the skin by direct inoculation.

Parasite.—There are three varieties of the parasite: tertian, quartan, and the parasite of estivo-autumnal fever.

Description of Parasites.—Tertian.—Its life's cycle is forty-eight hours. When first seen in the red blood-cell in its youngest form, it is a small hyaline ameboid body. It continues to grow at the expense of the erythrocyte, which rapidly loses its color, becoming quite pale. The parasite soon takes on fine pigment that is of a yellowish-brown color. The ameboid movement is quite active, and the pigment also appears to be in rapid motion. As it reaches maturity the erythrocyte increases in size, and when full grown, is larger than the normal red blood-cell. The pigment now gathers to the center and segmentation occurs, forming a roset body; there are usually two rows of leaflets, each segment forming into a distinct spore, numbering from twelve to twenty.

The shell of the corpuscle ruptures, and the spores are liberated about the time of the paroxysm. These again attempt to gain entrance to the healthy red blood-cells, the phagocytes, however, destroying the greater number. Flagellate bodies may be regarded as accidental forms developed from the full-grown parasite.

Quartan.—The cycle of development in this variety is seventy-two hours. In the red blood-cell in its youngest form it is found as a small hyaline body. Its growth is less rapid than the variety just described, and takes on pigment that is quite coarse and of a dark-brown color. As the parasite grows the erythrocytes decrease in size and become brassy in color. Ameboid movement in the quartan type of parasite is sluggish, and the pigment is not in active motion; when full grown, it is smaller than the red blood-cell. The pigment now gathers to the center as a star-shaped mass and segmentation occurs, forming one row of leaflets that develop in from

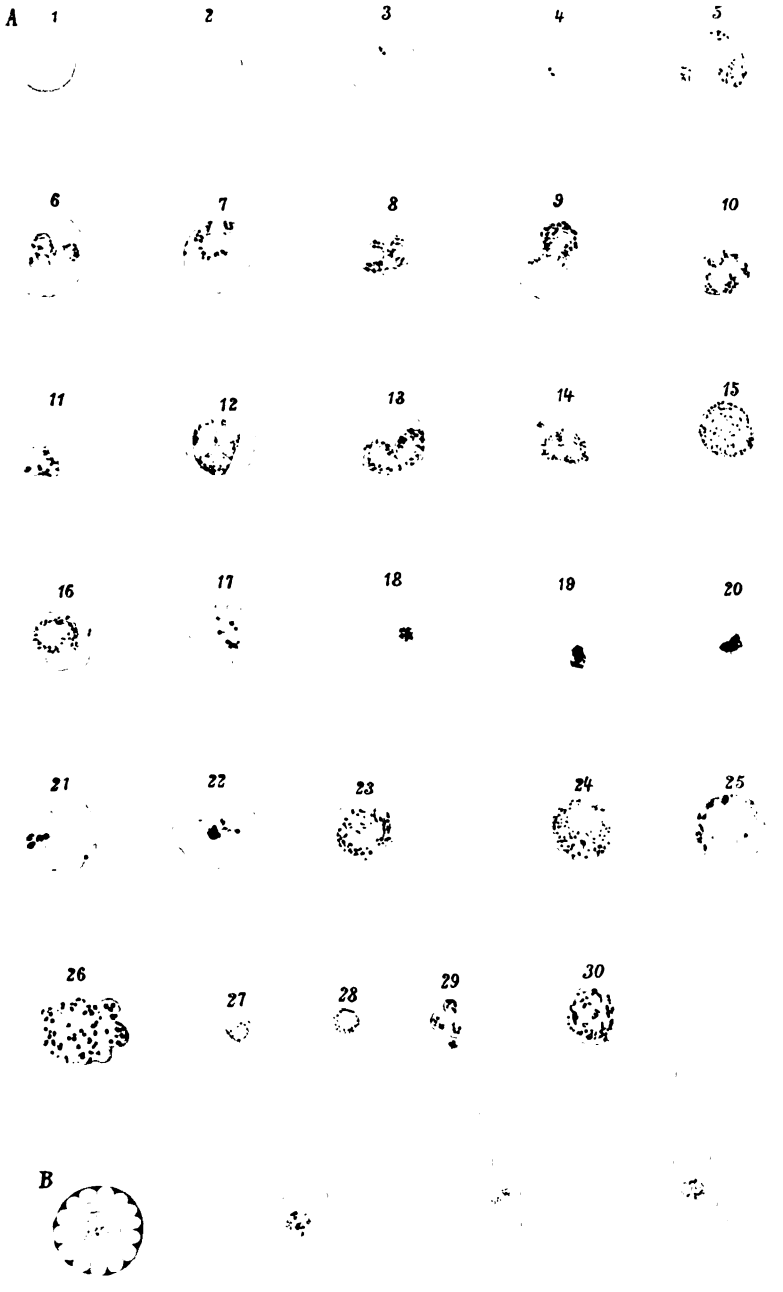
Description for Plate V.

A. Figs. 1 to 22.—Stages of development of the tertian parasite. (Fig. 17 and Fig. 18, after Thayer and Hewetson).

Figs. 23 to 29.—Hydropic degenerated malarial bodies.

B. Schematic sporulation of the tertian parasite (after Golgi). (From Mannaberg, "Die Malaria Krankheiten.")

Plate V.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Description for Plate VI.

A. Figs. 1 to 22.—Stages of development of the quartan parasite.

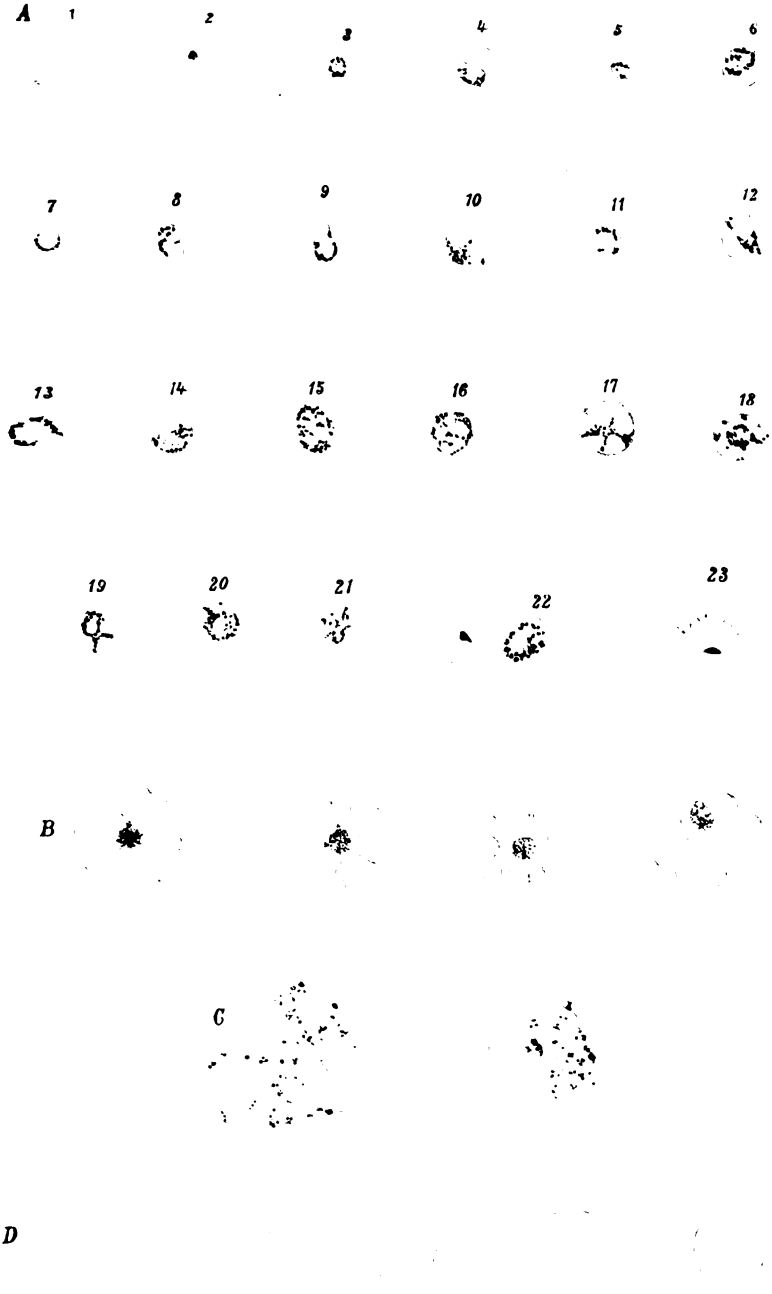
Fig. 23.—Rare sporulation form (after Canalis).

B. Schematic sporulation of the quartan parasite (after Golgi).

C. Leukocytes containing melanin.

D. Various vacuolation of the erythrocytes. (From Mannaberg, "Die Malaria Krankheiten.")

Plate VI.



Description for Plate VII.

A. Figs. 1 to 6.—Pigmented quotidian parasites.

B. Figs. 7 to 13.—Unpigmented quotidian parasites.

C. Figs. 14 to 19.—Malignant tertian parasites.

D. Fig. 20.—Brass-colored erythrocyte.

Figs. 21 to 37.—Crescentic bodies.

Figs. 24 to 26.—Fusion of two ameboid parasites (copulation).

Fig. 27.—Conjunction of two bodies.

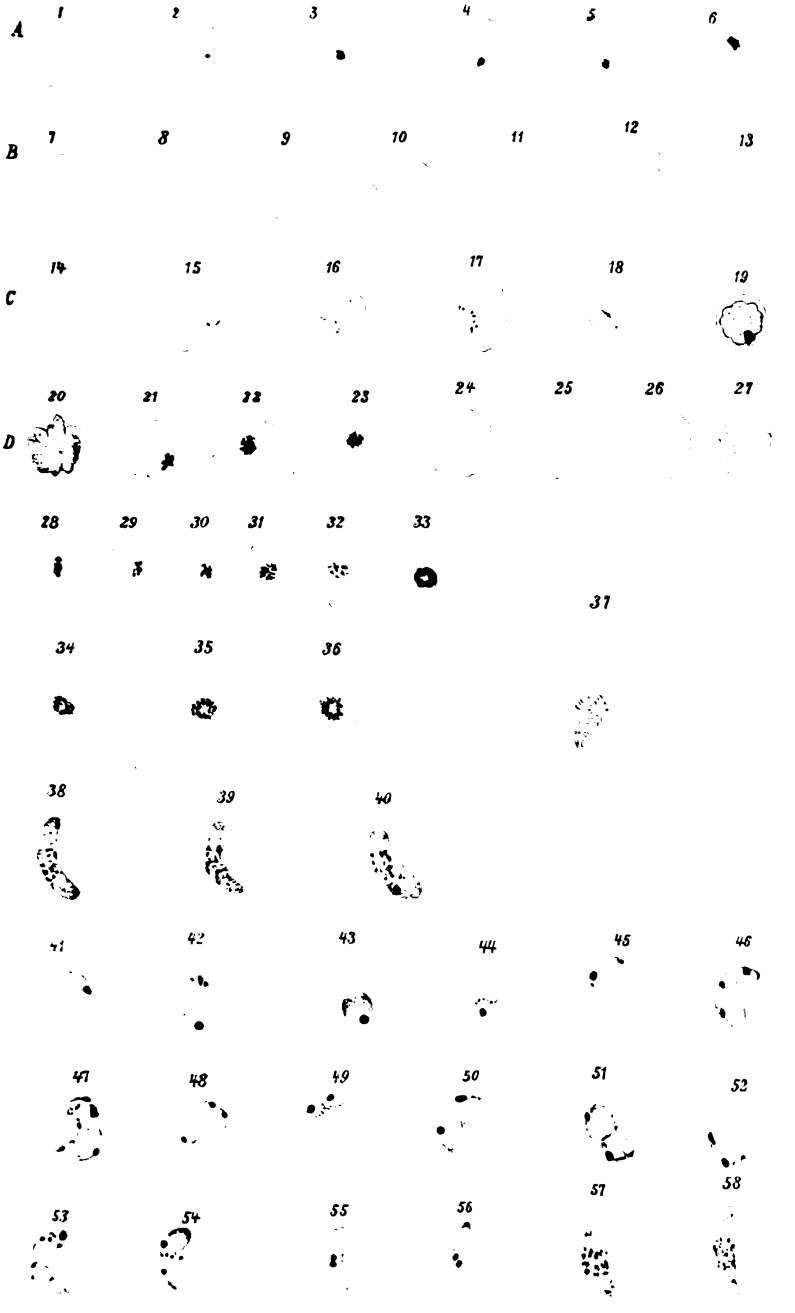
Figs. 38 to 40.—Stained crescents (by Romanowsky's method).

Figs. 41 to 58.—Stained parasites of the second group.

Figs. 49 to 57.—Formation of the crescents (conjunction of the bodies) (stained with hematoxylin after fixation by picric acid). (From Mannaberg, "Die Malaria Krankheiten.")

Note.—Mannaberg recognizes three varieties (namely, the pigmented quotidian, the npigmented quotidian, and the malignant tertian) of that class of parasites which is characterized by almost exclusive sporulation in the internal organs and the formation of crescentic bodies, described on page 199 as the estivo-autumnal parasite, without special subdivision.

Plate VII.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six to twelve spores. About the time of liberation of these spores the paroxysm occurs. Flagellate bodies may also be found.

Estivo-autumnal.—The youngest form of this parasite is found in the red blood-cell as a hyaline ameboid body, being usually ring-shaped and appearing toward the periphery of the corpuscle. The life cycle of this parasite varies from twenty-four to forty-eight hours, and occasionally longer. The hyaline bodies are found in the peripheral circulation; further maturity of the organism takes place in the internal organs—spleen, liver, bone-marrow, etc. By splenic puncture various stages of the development may easily be found. After the infection has persisted for from five to seven days, crescent-shaped bodies and oval and round forms are found in the peripheral circulation as well as the hyaline bodies.

Flagellate bodies may also be found. (For method of examination of the blood-cells see p. 150.)

Period of Incubation.—Tertian parasite, ten days; quartan parasite, thirteen days; estivo-autumnal parasite, three days. (This represents the time required for a sufficient multiplication of the parasite to produce symptoms.)

The effects upon the system are probably produced by three conditions: first, changes resulting from disintegration of the erythrocyte and liberation of spores; second, accumulation of pigment from this disintegration; and third, the influence of toxic materials produced by the parasite.

Pathology.—The spleen is always enlarged and dark in color. In all cases of chronic malaria the spleen may show marked pigmentation of a slate color, and becomes greatly increased in size. Sometimes perisplenitis exists. Rarely, cirrhosis of the liver follows, but this organ reveals pigmentation. The kidneys are enlarged and of a reddish-gray color, and may show parenchymatous change. Considerable pigment is found. Bone-marrow is usually of a deep-red color, and may also show melanotic pigmentation. Amyloid disease may result from malaria. Anemia rapidly develops, as a result of malarial infection. The erythrocytes are reduced, in severe cases the number reaching 500,000 in a cubic millimeter. The hemoglobin shows a greater reduction than the corpuscles. Free pigment may be found in the plasma and also in the colorless corpuscles. The leukocytes are not increased. Malarial bodies are commonly found in the phagocytes. The long-continued attacks of malaria may give rise to malarial cachexia,

being severe anemia with jaundice. The complexion in this condition is muddy and of a grayish-yellow tint.

Symptomatology.—Tertian.—*Prodromes* are rare, and are characterized by periodicity, consisting of vague pains, slight nausea, and chilliness. The paroxysm consists of three stages: *chill*, *fever*, and *sweating*.

First Stage.—A few hours before the chill headache and a sense of uneasiness may be experienced. The onset is abrupt, with chilliness resulting in a distinct rigor; this lasts from fifteen to forty minutes or more. The symptoms accom-

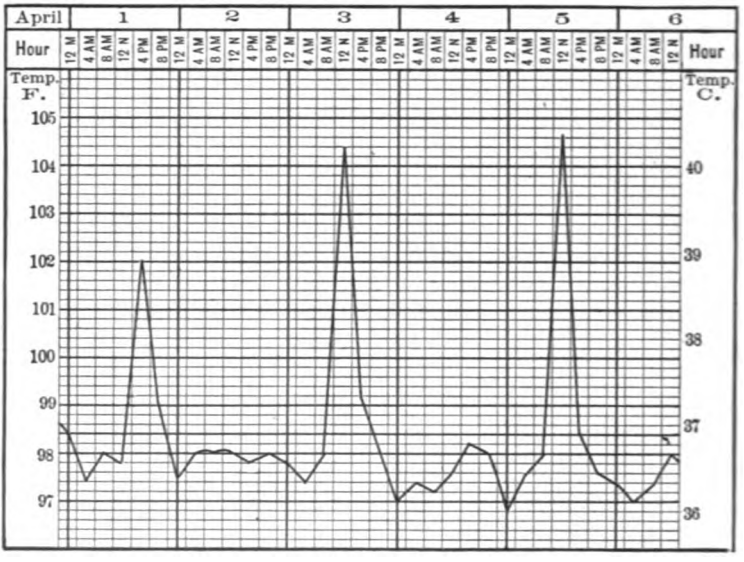


Fig. 28.—Temperature-curve in a case of tertian fever.

panying this stage are creepy sensations over the body, especially down the spine, yawning, and a certain amount of cyanosis. These symptoms are evidences of the contraction of the peripheral blood-vessels, causing absence of blood in the periphery of the body, with an increase in the visceral blood. Occasionally, there is nausea; sometimes there are vomiting, chattering of the teeth, and a rapid pulse, which is small and hard. The temperature, as registered by a surface thermometer, is subnormal, but the rectal and axillary temperature is high.

Second Stage.—The cold stage merges into the hot stage, which lasts from one to four hours. There are now sensations of heat, flushed face, injected eyes, full, bounding pulse, thirst, intense headache, restlessness, and even delirium. The temperature rises to 103° F. or higher.

Third Stage.—The hot stage is soon followed by the third stage—that of sweating; this begins with sweating of the face, rapidly spreading over the entire body, terminating in a very profuse sweat, lasting from one to three hours. The temperature begins to fall, and at the end of this stage reaches the normal or subnormal. Headache persists for some time afterward, as a rule being frontal in character. Herpes is frequently seen, and albuminuria may be present.

Intermission.—This is followed by a period of intermission lasting from thirty-six to forty-eight hours, during which time the temperature is normal or subnormal. The patient feels perfectly well, and is able to be about. This period continues until the next paroxysm, when the train of symptoms is again repeated. The regularity of these stages depends upon the uniform growth of the parasite, so that they sporulate almost simultaneously.

Occasionally, an individual is infected upon two successive days. In such an event there are two distinct groups of parasites that sporulate on consecutive days, causing a daily paroxysm (quotidian fever). In long-continued infections the growth of the parasite may become somewhat irregular, so that segmentation does not take place at a given time. As a result of this the chill may be prolonged or even absent. The fever may be somewhat irregular or, in rare cases, continued or absent. The sweating stage may show variation. In order to ascertain the variety of the type a blood examination is necessary.

Quartan Type.—This is the rarest of the malarial infections. It is found in parts of Italy, especially the Pontine marshes. The clinical manifestations of the paroxysms are similar to those just described in the tertian variety. The paroxysm occurs every seventy-two hours in the single infection. A double infection may occur, giving rise to a paroxysm on two successive days; then a day intervenes, and again two successive paroxysms follow, etc. The quotidian type of fever may also occur as the result of a triple infection.

ESTIVO-AUTUMNAL TYPE.

(SOMETIMES CALLED CONTINUED, REMITTENT, OR IRREGULAR.)

Varieties.—First, quotidian intermittent fever; second, tertian estivo-autumnal fever; third, pernicious estivo-autumnal malaria.

Estivo-autumnal fever is commonly found in the tropics, and rarely in temperate climates.

Quotidian Intermittent.—This is characterized by daily paroxysms, as described in the tertian variety. The regularity, however, has a tendency to vary.

Tertian Type.—This is characterized by paroxysms every

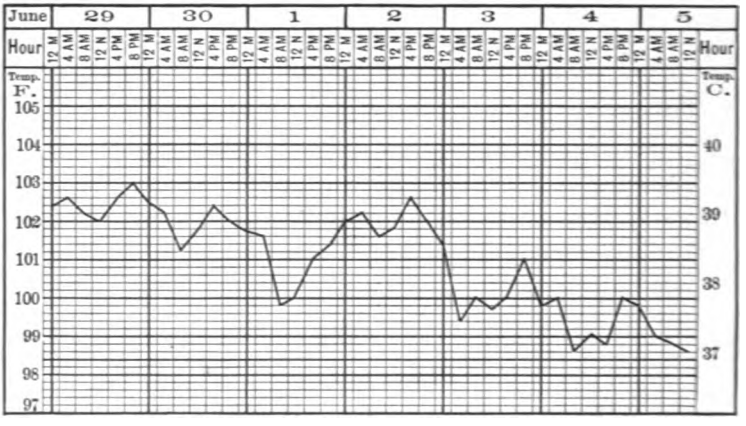


Fig. 29.—Temperature-curve in a case of estivo-autumnal fever.

forty-eight hours that may resemble the tertian type, and show a tendency to become irregular.

Pernicious Malaria.—This type is commonly associated with the estivo-autumnal parasite—rarely with the tertian. It is produced either as a result of the severe infection, usually, of the estivo-autumnal parasite or of great abundance of parasites.

Different Groups.—Three distinct and well-defined groups have been differentiated: the algid, the comatose, and the hemorrhagic form.

Algid Form.—The algid form is characterized by marked gastro-intestinal symptoms, vomiting, abdominal cramp, with

frequent stools. The urine is diminished, the pulse is feeble, and the temperature may be normal or subnormal.

Comatose Form.—The comatose form closely resembles an apoplectic stroke. The subject is suddenly stricken with unconsciousness or with acute delirium, with rapidly oncoming coma and chill. The unconsciousness continues, and the patient dies in coma. The temperature may be elevated at first, but in a day or two it falls to normal or subnormal.

Hemorrhagic Form.—The hemorrhagic type may occur in all the forms of severe malarial infections. Hemorrhage occurs most frequently from the kidneys, being either a hemoglobinuria or a hematuria. Suppression of the urine is occasionally met with, the patient dying from this cause. It is often called "bilious hemorrhagic fever."

Malarial Cachexia.—It is the result of long-standing infection or repeated infections, causing severe anemia and characteristic discoloration of the skin, being of a muddy grayish color. The enlargement of the spleen is marked.

Combination of Type.—Some of the varieties just described may be combined.

Complications and Sequels.—Pneumonia, dysentery, nephritis, and enteric fever are sometimes concurrent affections. To the latter cases the term typhomalarial fever has been incorrectly applied. There is no mixture of type, the enteric fever running its course, the malaria being an intercurrent affection, or vice versâ. Other complications, such as tuberculosis, orchitis, or adenitis, may occur.

Relapses.—Relapses are frequent, occurring after a period of weeks or months, sometimes of a year or more.

Diagnosis.—Diagnosis depends upon the finding of the parasites in the blood, and it may be said with accuracy that the type of fever may be determined, and with some certainty the time of the paroxysms. No diagnosis of malarial fever should be made in any fever showing irregular or regular type without the presence of the plasmodium. The therapeutic test should not be employed as a method of diagnosis.

Prognosis.—In temperate climates the prognosis is good, the mortality being very low. In tropic and subtropic countries in which the severer infections by the estivo-autumnal parasites occur, the prognosis is unfavorable unless quinin can be administered early and in sufficient quantities.

Prophylaxis.—Thorough drainage and systematic cultivation decreases its prevalence. If one enter into a malarious

district, high ground should be selected for living purposes and the night air avoided. Mosquito-netting is of use. Quinin may be administered in small doses as a prophylactic.

Routine Treatment.—Rest in bed, if possible, is desirable, especially in the severer infections. Quinin is the specific, and should be administered in doses sufficient to produce its physiologic effect. In the ordinary tertian form fifteen to twenty grains a day, preferably given in solution, will be usually found sufficient. In the severer forms and in pernicious malarial fever it is always best to give quinin hypodermically. The treatment may be begun by the preliminary administration of a laxative dose of calomel, but the specific (quinin) should be employed as soon as possible. Arsenic and methylene-blue may be used. A spontaneous recovery rarely takes place.

Treatment in any case should be continued for a period of some weeks after all symptoms have disappeared, as relapses are common.

Treatment of Malarial Cachexia.—Change of climate, a long sea voyage, and arsenic constitute the most favorable treatment of this condition. Quinin is of very little service. Warburg's tincture is often useful.

THE EXANTHEMATA OR ERUPTIVE FEVERS.

This group comprises scarlet fever, measles, German measles, variola, vaccinia, varicella, and, for the sake of convenience, erysipelas may be included. These fevers have certain well-marked characteristics. They are diseases of childhood, and are eminently contagious, having a known and well-defined period of incubation. They are all self-limited. Each is characterized by a well-defined eruption, appearing upon a certain day and in a definite manner. They have special sequels. There is no specific treatment, except in the case of vaccination, which is preventive in the variolous diseases.

SCARLET FEVER.

Definition.—An acute, specific, contagious disease, characterized by a scarlet eruption that occurs early on the second day, with sore throat, high fever, and frequent implication of the kidney.

Synonym.—Scarlatina.

This disease was first clearly described and differentiated from measles by Sydenham some time between the years 1661 and 1665. It first made its appearance in America in 1735. It prevails in all parts of the world, but is more common in cold and temperate climates. When it occurs in the tropics, it is comparatively mild.

Etiology.—Predisposing Causes.—Age is an important predisposing cause: it rarely occurs after the tenth year of life, although no age may be said to be exempt. Neither sex nor occupation predisposes. The form of so-called scarlet fever taking place after surgical operations, or coming under the hands of the obstetrician, may be due directly to infection. Surgical scarlatina has become a comparatively rare disease.

Climate is of some importance, the disease existing principally in cold and temperate regions. Epidemics are more prevalent in the winter. Sporadic cases may appear at any time. There is a marked personal predisposition, some members of a family being more liable to the disease than others. A certain proportion of the population is particularly immune, even failing to contract the disease when directly exposed.

Exciting Cause.—Undoubtedly a specific, infectious principle; it is always derived from a previous case. The exciting cause has not yet been demonstrated; the infection is readily carried by fomites. The vitality of the infectious principle may be measured by months or by a year or more; it is extremely tenacious. The poison leaves the body largely by the scales that are shed during the period of desquamation, and probably by exhalation. The poison may also leave the body by the urine and by pus from abscesses. The disease is contagious throughout its entire course, — probably more so during the time that the rash makes its appearance,— and persists until desquamation has been entirely completed, or even longer in cases attended by discharges from ulcerated surfaces or abscesses. The infective principle probably enters the body by the respiratory mucous membrane and the digestive tract. One attack confers immunity, as a rule.

Pathology.— The toxic agent produces inflammation of the tonsils and adjacent parts, causing fever and a diffuse rash. There are no characteristic changes produced in the organs or the tissues. Morbid conditions occurring in the intense febrile processes are found in this disease. Enlargement of the lymph-glands in various parts of the body, and

complications, such as endocarditis, pericarditis, and inflammatory changes in the kidney (postscarlatinal nephritis) are common.

Period of Incubation.—The period of incubation is from four to seven days.

Symptoms.—The disease appears abruptly, with a chill, high fever, and headache. In children convulsions and vomiting are common. Vomiting is, perhaps, a more characteristic symptom in this fever than in any other of the acute infectious diseases occurring in children. Sore throat is an early manifestation, consisting of an intense hyperemia of the pharynx, the half-arches, and the tonsils. The fever is high from the onset,—103° F. to 105° F.,—and even rises with the appearance of the eruption, which may be at the end of the first or the beginning of the second day; it remains high for four or five days, then falls in uncomplicated cases by rapid lysis.

Eruption.—The eruption consists of pinhead points of a deep-red color, appearing first upon the neck and chest and spreading rapidly all over the body except certain parts of the face, the mouth, and chin. These red points, which are close together, soon coalesce, giving a diffuse pinkish or reddish appearance to the entire skin, which presents a boiled-lobster color. With this intense inflammatory process, which is a true dermatitis, some slight edema may be noted. Throughout the entire eruption raised papules are found. Itching during this time is a prominent symptom. The eruption lasts about four or five days.

Throat Symptoms.—During the time that the eruption appears and throughout its course sore throat is a prominent symptom, even in the mildest cases. In severe cases diphtheric exudates are extremely likely to occur.

The tongue in this disease is characteristic: a white fur soon makes its appearance, which in a day or two peels off, leaving raised papillæ that give the tongue the appearance of a raspberry or strawberry; this is sometimes called the "cat tongue."

Nervous Symptoms.—Nervous symptoms are prominent: headache, delirium, and coma, and in children convulsions may mark the onset.

Special Symptoms.—The pulse-rate is increased in frequency —in mild cases from 120 to 140, and higher in the severer cases. Hemic murmurs are heard at the base of the heart. Slight enlargement of the spleen may be noticed. The urine

presents the usual characteristics of febrile urine, being scanty, high colored, and of high specific gravity, with albuminuria (toxic). The lymph-glands of the neck become prominent and swollen.

Defervescence.—Defervescence occurs in from two to four days after the exanthem has been prominent. The fever and sore throat decline as the eruption begins to fade.

Desquamation.—The eruption first begins to disappear at the point at which it made its earliest appearance. The skin peels off, usually in large scales, occasionally in fine ones. Entire casts of the hand or foot may come away during this stage. The duration of the period of desquamation is a variable one, and it may last from three or four days to a month or more.

During the period of desquamation polyarthritis and inflammation of the tendons may appear. The joint inflammation is generally of a fleeting character, and does not persist, excepting in those cases in which pus forms and the joint becomes septic. A common occurrence during this period is acute nephritis, known as *postscarlatinal nephritis.*

Nephritis.—The symptoms of this important condition are ushered in by a rise in temperature. The face becomes edematous, edema also showing itself in the feet, with extreme pallor of the face. The urine is scanty, and anuria may even occur; it may also contain blood. Chemic and microscopic examination of the urine shows all the characteristics of an acute nephritis.

Eye Symptoms.—Keratitis and iritis are met with.

Ear Symptoms.—Otitis media is very common in this affection, appearing more frequently than in any of the other acute infectious diseases.

Gastro-intestinal Tract.—The stomach and intestines may show symptoms of inflammation, or hemorrhages may occur. At the onset of the disease diarrhea frequently appears, soon, however, giving place to constipation.

Respiratory Symptoms.—Symptoms relating to the respiratory system are rare in scarlatina.

From the general infective process, especially in the severer forms, septicemia and pyemia occur.

Complications.—Diphtheria is the most important complication, and occurs in nearly all severe cases. Endocarditis appears; less commonly, pericarditis. Meningitis and peri-

tonitis complicate this disease. Pleurisy with and without effusion is not rare.

Sequels.—Enlarged lymphatic glands, chronic joint affections (with and without pus), hemorrhagic diathesis, monoplegia, hemiplegia, peripheral neuritis, hysteria, and anemia are the common sequels.

Varieties.—Four well-defined varieties of this disease are recognized: (1) Scarlatina simplex, or simple scarlatina; (2) scarlatina anginosa, or the variety in which the throat symptoms are most prominent; (3) scarlatina maligna (hemorrhagic scarlet fever); (4) latent, larval, or undeveloped scarlatina.

Scarlatina Simplex.—The symptoms are those already described, in which few, if any, complications occur, the active symptoms being over in a week or less.

Scarlatina Anginosa.—Scarlatina anginosa is characterized by prominence of throat symptoms, diphtheria being an almost constant complication. Great enlargement of the lymphatic glands of the neck shows itself in this form. The appearance is characteristic, and has been called the "collar of brawn."

Scarlatina Maligna.—Scarlatina maligna shows all the symptoms of an intense infection. The eruption may appear as petechiæ; the fever is high, hyperpyrexia being common; and the disease may end fatally in two or three days, with all the symptoms of an intense toxemia.

Scarlatina Latens.—In this form the symptoms are mild; fever may be moderate, the eruption appearing scantily or not at all, and may only be recognized by complications or sequels, particularly postscarlatinal nephritis.

Diagnosis.—This depends upon the knowledge of an epidemic; the sudden onset, with chill or convulsions, vomiting, high temperature, headache, sore throat, an exceedingly rapid pulse,—140 or more,—and the appearance of the eruption late in the first or early in the second day.

Differential Diagnosis.—This must be made between scarlet fever, measles, German measles, and simple erythema. In simple erythema fever is usually absent, sore throat is not prominent, the eruption shows itself in different parts of the body and not as a general diffused rash, and there is an absence of desquamation. In doubtful cases some time must elapse before the diagnosis can be definitely made.

	Scarlet Fever.	*Measles.*	*Rubella.*
Period of incubation	Four to seven days.	About ten days.	About eighteen days.
Rash	Dusky red and diffuse; great itching of the skin appearing from first to second day.	Papular, brick-red, darker, crescentic in shape, occurring about the mouth and forehead, with intervening healthy skin; eruption on fourth day.	Rose-red spots, irregular in outline, appearing upon the first day.
Catarrhal symptoms	Throat affection prominent; conjunctivæ unaffected; lung complications rare; diarrhea prominent early.	Little or no sore throat; catarrh; conjunctivitis; lacrimation and photophobia; bronchitis marked; bronchopneumonia common; diarrhea frequent.	Slight sore throat; conjunctivitis; bronchitis slight; no diarrhea.
Lymphatic glands	Prominent about the neck, proportionate to throat implication.	Not marked early; limited to the angle of the jaw.	Generally enlarged and tender, the posterior chain being particularly implicated.
General symptoms	Characteristic (strawberry) tongue; high temperature; rapid pulse; vomiting; marked nervous symptoms.	Little depression; tongue furred; anorexia; temperature not so high; pulse in proportion to the fever; marked catarrhal symptoms.	Little or no depression; appetite often retained; temperature slightly above normal; little change in pulse; symptoms slight.
Albuminuria	Frequent.	Rare.	Absent.
Convalescence	Prolonged, owing to complications.	May be prolonged.	Rapid.
Desquamation	Copious, in large shreds.	Seldom copious; fine scales.	May be copious; always in fine shreds.

Prognosis.—In very young children—under five years—the prognosis is extremely grave. In older ones in whom the severer complications are absent recovery usually takes place. Diphtheria is one of the most serious complications, and renders the prognosis grave. Early severe nervous symptoms, high fever, persistent vomiting, and rapid pulse are unfavorable.

Treatment.—Strict isolation is important. A room should be selected, if possible, at the top of the house. All unnecessary articles of wearing apparel, bed-linen, etc., that have come into contact with the patient should be destroyed by fire. The room should have a temperature of about 70° F., with good ventilation. An open fireplace is extremely useful for this purpose. The scales during the period of desquamation should be carefully collected and burned.

Diet.—The diet should be of an easily assimilated, nutritious character. Fluids, such as pure drinking-water or the alkaline mineral waters, should be freely administered. Milk is a necessary article of diet.

There is no specific treatment. A mild laxative should be given at the onset. This should not, however, be continued throughout the course of the disease, on account of the inflammation that may be present in the bowels. Tepid spong-

ing at least twice daily is necessary in severe cases. The cold bath is not advisable; warm bathing is, however, usually very grateful to the patient.

During the period of eruption an animal fat used as an inunction to the skin prevents itching and hastens desquamation. The administration of chloral hydrate, as first recommended by J. C. Wilson, is of great use in the majority of cases. Sufficient should be given to the child to keep it in a mildly somnolent condition. It also acts as a diuretic, and in this way may prevent nephritis, which so frequently follows this disease.

It is necessary to make daily examinations of the urine during the period of desquamation so that nephritis may be detected as soon as possible.

Complications must be treated upon general principles.

MEASLES.

Definition.—An acute, infectious, contagious disease, characterized by marked catarrhal symptoms, especially of the respiratory tract, with a characteristic eruption occurring upon the fourth day.

Synonyms.—Morbilli; rubeola.

Etiology.—Particularly a disease of childhood. Sex not a predisposing cause. Occurring in temperate and cold climates, usually in the winter months.

Exciting Cause.—This is unknown, but is very probably a specific infecting principle that is carried by fomites and that is very diffusible.

Period of Incubation.—The period of incubation is about ten days. One attack usually confers immunity, but a second, a third, or even a fourth attack has been noted.

Pathology.—There is no characteristic lesion. The inflammatory areas in the skin show infiltration of leukocytes, especially into the vessels, the sebaceous glands, and the sweat-glands. Bronchopneumonia is commonly the cause of death in measles.

Symptoms.—The disease may begin with a chill, followed by fever that may reach 103° F., or higher, upon the first day, with catarrhal symptoms from the onset. Injected conjunctiva, lacrimation, photophobia, coryza, and some cough are early manifestations. Rales are heard in the chest. At this time bluish points, surrounded by a white area known as

Koplik's sign, may be noticed upon the buccal mucous membrane.

On the second day the temperature falls rapidly, and remains subfebrile for about two days. On or about the fourth day the characteristic eruption appears, with a rise in the temperature. The fever again reaches 103° F. to 104° F. The eruption appears first upon the face and neck, spreads rapidly over the entire body, from twelve to thirty-six hours usually elapsing before the whole body is covered. The eruption consists of a rose-red or brownish maculopapular eruption raised above the skin, *with intervening healthy skin*, often arranged in a crescentic shape, especially upon the forehead and wrists. The eruption remains at its height for about four days. The catarrhal symptoms meanwhile continue, and even slight exacerbations take place. At the end of this period—about the eighth day from the beginning of the disease—the eruption fades, first from the face, neck, and chest, and then from the rest of the body. A fine desquamation occurs at the end of this period. The temperature about the seventh or eighth day falls abruptly to normal or slightly below, and in the absence of complications remains normal.

Gastro-intestinal Symptoms.—The tongue is coated, but not characteristic. Anorexia is complete. Diarrhea may be present from the onset of the attack, for it is probable that the same catarrhal process takes place in the digestive tract. The catarrhal symptoms gradually decline, and about the twelfth day of the disease in uncomplicated cases convalescence is reached. This is usually rapid, the child speedily regaining its normal condition.

Complications.—Bronchopneumonia is the most important and serious complication of this disease. Pleurisy occurs in a fair proportion of cases. Endocarditis and pericarditis are rare, and inflammation of the kidneys is extremely infrequent. Hemorrhagic measles also occurs.

Sequels.—Otitis media, chronic bronchitis, and tuberculosis are the most frequent sequels.

Diagnosis.—Depends upon the sudden onset, marked catarrhal symptoms, appearance of the rash upon the fourth day, typical fever-curve, and the prevalence of an epidemic.

Differential Diagnosis.—German measles, or rubella, may be readily differentiated by an absence of marked catarrhal symptoms, by the fact that the rash occurs upon the first day, and by the milder course of the affection.

Treatment. — Prophylaxis. — Prophylaxis presents great difficulties, owing to the great diffusion and infectiousness of the disease, and to the fact that the diagnosis can not usually be made before the characteristic eruption appears. If there are several children in the family, it is just as well if they all contract the disease, as in adults it is a very much more serious affection and complications are more likely to occur.

There is no specific treatment, and in uncomplicated cases medicines are unnecessary; a mild laxative at the onset is useful. The entire treatment should be directed to the prevention of complications, especially of bronchopneumonia. When complications occur, they must be treated upon general principles.

RUBELLA.

Definition.—An acute, infectious, contagious disease, occurring in epidemics, characterized by enlargement of the superficial lymphatic glands of the neck, slight fever, and mild catarrhal symptoms.

Synonyms.—Rötheln; French measles; German measles; epidemic roseola.

Etiology.—This disease was described as a substantive affection early in the eighteenth century, and even up to the present time it has been regarded by some clinicians as related to measles and scarlet fever. It is, however, an independent affection, characterized by its own symptomatology and clinical course. It is a disease of childhood, occurring mostly before the fifth year and more particularly in temperate climates.

Exciting Cause.—The exciting cause is unknown. It is highly contagious, and readily transmitted from the sick to the well. One attack confers immunity from itself, *but not from measles or scarlet fever.*

Period of Incubation.—The period of incubation is from one to three weeks, usually estimated at eighteen days.

Symptoms.—The onset is sudden, and probably the first symptom noticed is the eruption, which occurs irregularly over the face, neck, chest, body, and limbs, varying in individual cases and different epidemics. This multiform eruption may resemble erythema, urticaria, and, in some cases, that of true measles or scarlet fever. It is never, however, arranged in the form of crescents. It may be confluent or diffuse, last-

ing from two to four days. Desquamation occurs in fine scales. The fever is irregular, and may be absent altogether. Slight sore throat occurs, but this condition is usually trifling. There is enlargement of the superficial lymphatics, especially the cervical and postcervical chains. Suppuration is rare. The tongue is furred, the appetite is lost, and the urine presents the characteristics of febrile urine. Albuminuria is extremely infrequent.

Complications.—Complications are uncommon, and are mostly found in cachectic or strumous children in foundling asylums or other large institutions for the young. Bronchitis and pneumonia are the most common.

Prognosis.—The mortality is extremely low, less than 4% of the cases proving fatal.

Treatment.—Expectant and symptomatic.

VARIOLA (SMALLPOX).

Definition.—An acute, infectious fever, markedly contagious, characterized by a typical eruption that passes through successive stages, and by high temperature and septic phenomena.

Etiology.—Smallpox has been known from the earliest ages, even mummies having been found presenting the characteristic scars. It was introduced into Europe by the Crusaders, and was the great scourge of that continent for centuries, until the immortal discovery by Jenner of vaccination. Neither sex nor age confers immunity. The fetus *in utero* may develop smallpox if the mother is affected. The liability to this disease is universal, except as controlled by vaccination.

Negroes are especially prone to this disease. Climate and season are without influence, but epidemics more commonly occur in winter.

Exciting Cause.—This is as yet unknown. The poison is rapidly communicable, both through the atmosphere and by fomites, and may retain its vitality for months and years. The disease is contagious from its onset. One attack usually confers immunity, but second attacks have occurred in isolated instances. It may be contracted from the cadaver of one having died from smallpox.

Pathology.—The pathology consists principally in the cutaneous lesion, the pock appearing first as a distinct macule

as a result of inflammation of the papillary layer of the skin, which causes the development of a hard papule. Liquefaction necrosis taking place, a clear serum exudates, and thus we have the conversion of the papule into a vesicle, and finally pus-corpuscles appear in the fluid, giving rise to the pustule. The umbilication is due either to a hair follicle in the center of the inflammatory zone, or to absorption being more rapid toward the center, thus causing the depression.

In the hemorrhagic variety a bloody exudation is found in the vesicle, and the inflammatory area is also deeper. If suppuration be marked, healing by granulation and cicatrization follows, and the deformity develops as a result of the contracting scar. Parenchymatous degeneration is found in the heart, liver, and kidneys. In severe cases fatty degeneration of the liver may be present. Hemorrhages may occur from the serous surfaces and also into the tissues. The leukocytes show a marked increase during the period of suppuration, which is especially marked in the hemorrhagic variety.

In fatal cases the blood is dark, coagulation being imperfect, as is found in other infections. Intense congestion of the internal organs with ulceration of the mucous membranes is found. The internal organs show the changes due to complications. The gastro-intestinal mucous membranes manifest the presence of the lesion in severe cases. The cutaneous lesions shrivel after death. Inflammations of parts of the brain and spinal cord are noted. In the hemorrhagic variety large extravasations of blood are found in the skin and in the mucous membranes. In malignant cases death usually occurs before the eruption makes its appearance.

Period of Incubation.—From ten to thirteen days.

Symptoms.—The stage of invasion is sudden, with a profound, protracted chill. Milder cases may develop only a slight chilliness, but even this may be prolonged for some hours. The temperature rises abruptly from 102° F. to 105° F.; the pulse is increased in rapidity from 120 to 130. General muscular pains are prominent, especially in the back, which are continuous and very severe. Headache is an early symptom. In children convulsions may occur, being followed by delirium and coma. These symptoms continue until about the third day, accompanied by nervous symptoms, such as restlessness and stupor; vomiting may also be present.

On or about the third day there is a sudden remission in the temperature, the pulse-rate falling, and the severe symp-

toms just enumerated suddenly disappearing, so that the patient imagines he is well. It will now be noticed that an eruption has appeared upon the face, thence spreading over the entire body. At first the rash is about the size of a pin-head, and soon becomes hard, feeling like a shot under the skin. The eruption may be *discrete* or *confluent*. This macular eruption is rapidly converted into a papule of a reddish color. There may be itching and burning attending these early eruptive symptoms.

In from twenty-four to forty-eight hours the rash has invaded the entire body. Great numbers of the individual

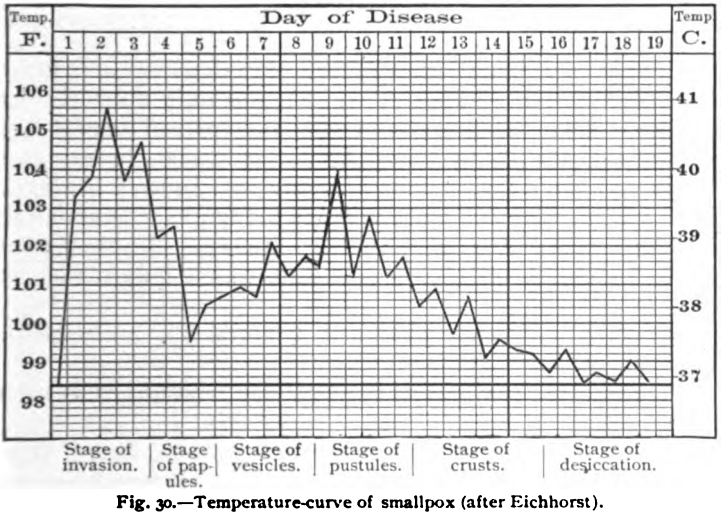


Fig. 30.—Temperature-curve of smallpox (after Eichhorst).

papules have appeared, these being irregular in size. The earlier ones soon become vesicular, this change taking place about the sixth or seventh day from the onset of the attack. In a day or so the fluid becomes turbid and purulent, the top being held down (*primary umbilication*); in from twenty-four to forty-eight hours this umbilication has disappeared, the pock now being conic in shape. About this time an intense red inflammatory areola is noticed about the base of the pock, and the eruption is now exceedingly painful. On or about the ninth day from the beginning of the disease suppuration begins in the pock, lasting about three days, when the apex of the cone drops in, due to the absorption of the contents of

the pock (*secondary umbilication*). When absorption has been completed, a crust forms that may remain for some days, these crusts falling off on or about the sixteenth day from the beginning of the disease, leaving depressed striated scars.

During the time of suppuration the temperature rises again, and may be higher than during the initial stage. New symptoms develop, which are septic in nature, such as convulsions, delirium, coma, chills, sweating, and diarrhea. The pulse is rapid and feeble; the urine is scanty and high colored, containing albumin, and the patient's condition is extremely grave. This fever, which is known as the *secondary fever*, continues as long as the suppurative process exists. About the time of suppuration a peculiar offensive odor is exuded from the body of the patient.

Preliminary Eruption.—A rash sometimes appears before the characteristic eruption of variola, generally upon the first or second day. This may resemble the rash of scarlet fever or measles, usually in triangular forms, which, having been discovered by Simon, are known as *Simon's triangles*. They appear upon the inner sides of the thighs, the abdomen, the upper part of the chest, and the forearm. In the hemorrhagic variety it occurs as petechiæ, which may remain for some time.

Varieties.—Discrete, confluent, hemorrhagic, and modified smallpox, or varioloid.

The **discrete form** shows the pustules separate, with intervening healthy skin, and is of a milder type.

In the **confluent form** the vesicles run together. They more rapidly become pustular, and all the symptoms are aggravated.

In the **hemorrhagic form** two varieties must be differentiated: (1) *Purpura variolosa*.—The eruption appears as petechiæ. No traces of the true pock can be seen. The skin becomes dusky, and purplish rashes appear all over the extremities. Hematuria may develop. The temperature may be low, but rises just before death. The body of the patient emits a terrible odor. The mind usually remains clear. This is the variety known as *black smallpox*. (2) The rash may occur in the usual way, but in the vesicular stage hemorrhages occur into the pock. Hemorrhages from other parts of the body are also common.

Complications and Sequels.—These are few in number, and are due to secondary infections. Inflammations and sup-

purative processes, such as abscess, furunculosis, and erysipelas, may occur. Diphtheric exudations may be found as a secondary affection. There may be bronchopneumonia and inflammation of the bones and cartilages.

Diagnosis.—It is difficult to confound any disease with variola when the rash is well developed. No other affection presents primary umbilication with consecutive changes from macula into papule, vesicle, pustule, crust, and scar. Previous to the appearance of the rash it may be impossible to diagnosticate this condition, but the knowledge of an epidemic should prevent blunders in diagnosis.

Prognosis.—This depends greatly upon vaccination. In unmodified smallpox the death-rate varies between 40% and 60%. One of the most important factors in the prognosis of variola vera (true smallpox) is age. At the extremes of life —in the very young and in the very old—it is an extremely fatal disease, the prognosis being more favorable between the ages of five and fifteen years. Sex is of no importance. In alcoholics and drunkards the mortality is extremely high, on account of the greater liability to the hemorrhagic or malignant varieties. Pregnant women almost invariably abort. The discrete variety is more favorable than the confluent. The hemorrhagic or malignant variety is almost invariably fatal.

The severity of the case depends greatly upon the amount of the eruption—the greater the eruption, the more serious the case. The appearance of the eruption has some weight in prognosis, as the more typical the eruption and the more regularly it goes through its successive stages, the more favorable is the prognosis.

Prophylaxis.—The prophylaxis against smallpox consists in *vaccination and revaccination.* When patients have been vaccinated or have had a previous attack of smallpox and complete immunity has not been obtained, they contract varioloid or modified smallpox when re-exposed to the disease.

VARIOLOID, OR VARIOLA MODIFICATA.

Definition.—This is smallpox modified by previous attack, vaccination, or inoculation. The proof consists in the fact that if an unprotected person be exposed to varioloid, he will contract variola vera and not variola modificata.

Symptoms.—The symptoms are usually the same as in unmodified smallpox, except that the initial symptoms are generally much milder. The eruption appears earlier,—usu-

ally within thirty-six hours,—and is not nearly so copious, there being, perhaps, but three or four, or sometimes from twenty to thirty, papules over the entire body.

Initial Rashes.—Initial rashes (Simon's triangles) occur oftener in varioloid than in variola. The eruption goes through the stages of macula, papule, and vesicle, rarely, if ever, reaching the pustular stage, and if pustules do form, they are very few in number; hence, as there is no pus to absorb, secondary fever does not take place and the grave symptoms of variola vera are averted.

Complications.—Complications rarely, if ever, occur in varioloid. All the symptoms are milder and the disease terminates sooner.

Treatment of Variolous Diseases.—Besides vaccination as a prophylaxis, complete isolation and disinfection are absolutely necessary. If the patient be treated at home, all unnecessary furniture and other articles should be removed from the sick-room. Everything coming in contact with the patient, such as wearing apparel, bed-linen, etc., should, if possible, be destroyed by fire. It must be remembered that the body of a smallpox patient is capable of transmitting the disease; therefore, after death the body should be wrapped in cloths that have been soaked in powerful disinfecting solutions, and should be removed as quickly as possible. Whenever practicable, *cremation* instead of burial should take place.

There is no specific treatment. Food, as a rule, is not well borne at first, and there may be much vomiting and diarrhea. Usually, acid drinks or small particles of ice are grateful to the patient. From the beginning of the disease detergent washes containing antiseptic solutions should be used for the mouth. Many drugs and methods have been used to prevent pitting. The most useful method for this purpose is that of covering the face with clean cloths saturated with warm water. These should be frequently renewed.

The room should be darkened: the solar light should be rigidly excluded. Iced applications should not be applied to any part of the body in which the eruption has appeared, as cold frequently prevents the pock from maturing. Warm baths two or three times daily are of decided benefit to the patient. During the time of secondary fever bold stimulation is necessary, as this is a septic process and must be treated upon these principles. If there should be great itching of the skin, with irritation, camphorated oil may be used. This has proved

beneficial in a number of cases. Attention should be given to all purulent discharges, which should be speedily removed, as their absorption means increased septic disturbance.

At the onset a laxative is useful, but this should not be repeated, as tendency to diarrhea exists. Pain should be overcome by small doses of opium, but care must be taken with this drug, as it tends to lock up the secretions. Restlessness and insomnia should be treated by trional and sulphonal. In the malignant varieties and in severe cases bold stimulation is necessary. In convalescence tonics are of service.

VACCINIA (COWPOX).

Definition.—A disease always conveyed by artificial inoculation from animal to man or from one human subject to another, having an eruption resembling smallpox, from which it is protective.

History.—To Sir Edward Jenner we owe this great discovery. In the year 1798 he first published the results of his experiments, and for many years his conclusions were questioned and ridiculed, but the process has now been almost universally adopted. As a result of this discovery, the spread and the mortality of smallpox have enormously diminished.

Whether or not vaccinia is a form of smallpox in animals, in which the symptoms are milder, is as yet a mooted question.

Vaccine Lymph.—Vaccine lymph consists of two kinds: that derived from the heifer, known as **animal** or **bovine lymph,** and that from the child, known as **humanized lymph.**

Pathology.—The lesion is limited to the part of the skin in which the vesicles develop. At the point of inoculation in which the vaccine virus is introduced the papillary layer of the derma becomes congested, and in from three to five days shows symptoms of slight inflammation, followed by an exudation of lymph. This increases, and as the layer of the epidermis is detached, another layer is lifted forward and forms the roof of the vesicle. The vesicle is not unilocular, as can readily be determined by pricking it, when it will be found that it does not collapse. It bears, in this respect, a striking resemblance to the vesicle of variola vera. The lymph is at first clear and thin, but soon becomes turbid and opaque. At this time a slight depression (umbilication) is seen at the

top of the vesicle. Desiccation now takes place, particularly at the center, and the pock dries up, forming a scab or crust. Upon removal of the scab a cicatrix is found beneath, which remains permanently. This cicatrix is peculiar, showing well-defined margins, being reticulated or foveated—at first red in color, in a short time becoming pale and permanent.

Symptoms of Vaccinia.—These are always produced by vaccination, and from two to three days after the introduction of the serum no symptoms are noticed except a slight scar, due to the instrument used in introducing the lymph. Upon the third or fourth day a faint redness appears around the point of inoculation, which gradually increases in extent until a distinct papule begins to make its appearance, becoming more and more prominent. Upon the fifth to the seventh day the papule changes to a vesicle, and the lymph that it contains is clear and transparent. In a day or two the lymph becomes turbid, pearly, and thicker.

On or about the eighth to ninth day the vesicle has reached its height, and is now turbid and yellowish in color, appearing as if it contained pus. At this time it is found to be distinctly umbilicated. Around the base of the vesicle a well-defined inflammatory zone makes its appearance, which becomes redder as it reaches the periphery. This is known as the inflammatory areola.

Constitutional Symptoms.—The constitutional symptoms noted at this time may be slight fever, anorexia, a feeling of general malaise, and pain under the arm in the axillary region. Upon careful examination it will be found that the axillary lymphatic glands are enlarged and painful. From the eleventh to the twelfth day the pock fades, becomes opaque, and desiccation is noticed at the center. The areola begins to fade, and upon the fifteenth day desiccation is complete, although the crust may not fall off until at the end of the third or fourth week. Upon the disappearance of the crust a distinct cicatrix is found beneath. This scar is at first red and pitted, showing radiating bands or striæ across it, and in a few weeks becomes pale and remains permanent.

Irregularities.—Irregularities sometimes occur in the development of the pock, and it is necessary to describe one of the most frequent, which is known as the "red-raspberry" excrescence. About a week after inoculation the papule appears, but instead of continuing to the stage of vesicle it remains hard and dense, assuming a bright-red color and

looking not unlike a small nevus. It is persistent, and may remain for weeks. There is no areola, and upon falling off, it leaves no scar. *This spurious form is not protective.*

Complications and Sequels.—These may be local or systemic, but the really serious ones are exceedingly rare. Inflammatory complications, which are due to trauma at the time of inoculation, may occur, such as a too extensive scarification. An abrasion of about one third of an inch in diameter will usually be found sufficient. Complications may occur from an injury to the pock after its development. Thus, if the roof of the pock is broken, an opening is formed through which secondary infection may take place. It is not wise to vaccinate in areas showing scrofular, tubercular, or eczematous skin affections, unless in the presence of exposure or an epidemic. The complications, as a rule, consist of lymphadenitis, phlegmons, profuse suppurative processes, dermatitis, etc., and with the secondary introduction of pyogenic bacteria may give rise to erysipelatous processes. Gangrene of the skin may occur, but this is rare, as are also septicemia and pyemia.

Vaccinal Syphilis.—Bovine lymph, which can now be produced in an absolutely pure state, should always be used. If humanized lymph must be employed, the physician should be careful to obtain it from a child with whose antecedents he is absolutely familiar. If a person who has been vaccinated subsequently develops syphilis, if the syphilis be due to vaccination, the primary sore or chancre must appear at the point of inoculation, otherwise it was not due to the vaccination.

Technic of Vaccination.—A simple thumb lancet is all that is necessary for this purpose. After it has been thoroughly sterilized, the surface at which the inoculation is to be practised should be cleansed with soap and water, then with alcohol, and allowed to dry. The skin may be prepared by removal of the epidermis through a series of scratches until the surface is abraded and a little exudation of serum occurs. *Avoid drawing blood.* Another method is to make four or five slight punctures under the skin, rubbing in the lymph, and working it beneath the edges of the epidermis by slight incisions. It is best always to make two points of inoculation. By this means the chance of success is doubled.

The point of selection for the vaccination is usually the outer surface of the arm, at or near the insertion of the deltoid. In case of a female child the vaccination may be practised on the leg, the point of inoculation being just below and at the outer

portion of the tibia. The left arm is usually preferred, because the child is mostly carried upon the left arm, in order that the right arm may be used for other purposes. Thus, the left arm of the child is free and the sore is not rubbed against the body of the person carrying it.

At the present time bovine virus is used almost exclusively.

As staphylococci are almost invariably present, it has been found that by mixing the lymph with sterilized glycerin (40%), sealing it up in tubes and putting it in ice-boxes for several weeks, these organisms are destroyed. The stables in which the cows are kept are rendered as clean as possible, all the animals being subjected to the tuberculin test. After the virus is obtained the animal is killed, and an autopsy is held to see that it is free from tuberculosis.

A healthy calf about six months old is chosen for the purpose. It is laid upon the table, and the abdomen and inner parts of the thighs are shaved and given an antiseptic toilet. A sterilized glycerinated virus is rubbed over the scarified place. The animal is well fed and watched for five days, then thoroughly cleaned again, and the crust or scab removed with a spoon curet. The mass is weighed and dried, ground up with sterilized glycerin, sealed in glass tubes, and placed in an ice-chest for five weeks. These glass tubes usually come ten in a box. The box also contains a rubber bulb or tube, which is attached to the glass after the ends are broken off, when it is desired to use the virus. The virus is obtained from the glass by squeezing the rubber tip or blowing through the rubber tube.

Humanized Virus.—Humanized virus is somewhat quicker in its action, and the constitutional symptoms are said to be milder. It is desirable not to vaccinate a child before the third month after birth, for if it be syphilitic, the symptoms will have shown themselves by this time.

Every one is susceptible to vaccinia, although this varies in individual cases. Vaccination should be practised and repractised until a typical scar results. A child should be vaccinated, as stated, at three months after birth, again at the end of seven years, and again at puberty, *and always after exposure to and in the presence of an epidemic of smallpox.*

VARICELLA.

Definition.—An acute, infectious, contagious disease of childhood, characterized by a vesicular eruption and mild constitutional disturbances. The disease is highly contagious.

Synonym.—Chickenpox.

Etiology.—This disease bears no relation to variola, and one attack while conferring immunity from itself, *does not* protect from *smallpox.*

Exciting Cause.—Not known. The poison is transmitted from the sick to the well by contact, and to a short distance through the air, and is carried by fomities. The disease usually occurs in epidemics. Sporadic cases are rare.

Predisposing Cause.—Age is the principal predisposing cause. It is most common between the fifth and tenth years of life, and is comparatively rare in adults.

Period of Incubation.—The period of incubation is usually given as from ten to fifteen days. It may be slightly longer or shorter than this.

Symptoms.—Prodromes are absent as a rule. The disease is ushered in by a mild chill. The appearance of the eruption marks the beginning of the disease. The exanthem shows itself as a small reddish point or papule, which in a very few hours becomes a vesicle. It is slightly elevated above the skin, rather than having the appearance of being under the skin. The vesicles are thin and transparent, and from one-eighth to one-fourth of an inch in diameter. The contents are at first clear and transparent. There is usually no areola. In the course of a few hours the vesicle becomes milky and begins to shrivel, with a depression at the top, from absorption of its contents (secondary umbilication), corresponding to the same condition in smallpox. This results as a yellowish-brown crust that, in about ten days from the beginning of the attack, and even before this, separates, leaving a more or less well-defined scar, which, in some cases, especially upon the face, remains permanently. The pox may appear upon the face, neck, scalp, wrists, and some parts of the body. It may be generalized and then found particularly upon the trunk. It may appear upon the cheek, tongue, palate, and even upon the conjunctiva and, occasionally, upon the genital organs. Constitutional symptoms are usually absent, and when present, are of an exceedingly mild type. Occasionally, the superficial

lymphatics may be enlarged. If the vesicles are scratched or injured, they become painful.

Complications.—Complications are rare. Occasionally, erysipelas results from secondary infection.

Diagnosis.—The diagnosis depends upon the appearance of the eruption upon the first day, with or without mild constitutional symptoms.

Differential Diagnosis.—Occasionally, mild grades of varioloid may be mistaken for chickenpox. In doubtful cases occurring in adults the scars of vaccination should be looked for. If well-defined scars be present, varioloid may be excluded. In varioloid the eruption does not come out until after from thirty-six to forty-eight hours of mild constitutional symptoms. In varicella the eruption marks the first manifestations of the disease. The knowledge of an epidemic should be important in differentiating the two affections.

Prognosis.—Almost invariably favorable.

Treatment.—Patient should be put to bed and given a bland diet. A gentle purge at the onset is of use. The lesions on the face should have protection from pricking and scratching: several layers of collodion may be painted over them for this purpose. In young children the hands should be enveloped in mittens or bandages. Isolation is not necessary, nor is disinfection, except by fresh air, as the disease is insignificant and so contagious that it is better for a person to have it in childhood than in adult life, when the disease is likely to be severer.

ERYSIPELAS.

Definition.—An acute, febrile, contagious disease, characterized by inflammation of the skin, with constitutional symptoms.

Synonyms.—St. Anthony's fire; "the rose."

Etiology.—One attack does not confer immunity, but rather predisposes to other attacks. It rarely occurs in epidemics. It may appear as a local inflammation without constitutional implication. It is placed in the group of eruptive diseases on account of its having a specific eruption with constitutional symptoms and running a definite course.

Predisposing Causes.—Occurs at all seasons of the year and in all parts of the world. Sex has no influence. It may occur at any period of life, but is especially liable to develop in debilitated and cachectic persons and in the course of chronic

pulmonary tuberculosis. Modern pathology teaches that erysipelas is due to the entrance into the organism of toxic agents that produce inflammatory conditions of the skin through an open wound; hence this disease is liable to occur in the puerperal state. It has sometimes resulted from a small crack in the skin at the angle of the nose or at the mouth, or from a scratch upon the face; from the piercing of the ears for earrings or from the ulceration about a carious tooth. In some instances the point of entrance of the specific agent can not be found.

Exciting Cause.—This disease is due to the streptococcus erysipelatos of Fehleisen. (For a description of the germ see p. 101.) Since the discovery of this germ the division into idiopathic erysipelas must be entirely abandoned.

It has not been shown to be transmissible by the air, having occurred without obvious traumatism, abrasions, or inoculations, and may enter the organism by the respiratory surfaces. Most cases occur in the spring of the year. Occasionally, a house epidemic has occurred.

Pathology.—When the specific germ gains entrance into the tissues, it produces the phenomena of severe inflammation. These changes may extend to the subcutaneous tissues. Occasionally, the inflammatory changes will go on to the point of suppuration. The inflammatory process is usually circumscribed. Postmortem appearance will reveal, besides the local lesion, evidences of granular degeneration of the internal organs, such as the heart, kidney, and liver. These changes are, as a rule, produced by the toxins, the organism rarely gaining access to the blood-corpuscles. In such an event suppurative changes are produced. A marked leukocytosis of an inflammatory character is present.

Period of Incubation.—From three to seven days; in artificial inoculation, from fifteen to sixty hours.

Symptoms.—The disease usually begins with a chill, which may be mild or severe, and is followed by the development of the eruption. With this there are usually gastric distress and some febrile reaction (the temperature not being characteristic), with the signs of an irritative, itchy, swollen skin at the point at which the eruption begins. There are heat, tension, and burning in the part. The eruption shows a decided elevation with a distinct prominent margin, red and puffy in the center. It is irregularly circumscribed, and there is an abrupt descent to the level of the surrounding skin. The

involved area is discolored, of a bright crimson color, and glossy in appearance.

The skin is hot and tender, but firm and smooth. For two or three days the area extends uniformly but irregularly; the margins, however, being always abrupt, well-defined, and circumscribed. In mild cases, after two or three days the eruption may become stationary and the process undergo resolution, with a remission in the fever, desquamation of the inflamed area, subsidence of the edema, and the color changing from a bright red to a bluish purple or light brown. The desquamation occurs in scaly masses. In mild cases the eruption may show no tendency to spread, but may remain where it first appeared, involving the whole face or side of the scalp, and terminating in recovery in from two to three weeks. In the severer cases the inflammation spreads over wider surfaces and invades the adjacent skin, leaving the portions in the center pale and red and undergoing desquamation.

If bullæ are formed, serum is thrown out under the epidermis, or if the inflammation has been severe, true blisters occur. The fluid in such cases is limpid, of a straw color, and may be purulent. As resolution takes place crusts are formed that gradually break down. In malignant cases areas of gangrene form, the skin repairing by sloughing and cicatrization. If the erysipelas travels over a considerable area, disappearing at one point and appearing at another, it is known as "*erysipelas ambulans*," or "*wandering erysipelas.*" This is much more serious, and the affection is likely to terminate fatally, although the process may be prolonged over a series of weeks.

In severe cases in which the affection appears upon the face the eyes are closed, the lips project, the ears are shapeless and cushiony, the nose is deformed, the cheeks encroaching upon it, and secretions may collect at the corners of the eyes, mouth, or nose. The whole face is painfully distorted and deformed. The tongue is coated with a yellowish fur, becoming dry and glazed and of a reddish hue. In severe cases marked nervous symptoms develop, delirium, coma, subsultus tendinum, and carphology, and the temperature may rise to 106° F. or higher.

In fatal cases hemorrhages take place in the blebs upon the skin, and gangrene makes its appearance. This may occur in infants, in the aged, in those subject to chronic alcoholism, and in cachectic individuals.

The disease commonly starts from the point of the ear, tip

of the nose, from a point of vaccination, or from ulcers upon the lower extremities. The bowels are usually constipated. The urine has the character of febrile urine, and even in mild cases true albuminuria occurs early in the course of the disease.

Surgical Erysipelas.—Surgical erysipelas rarely occurs in these days of antiseptic methods.

Complications and Sequels.—Complications and sequels are not numerous. Albuminuria occurs in serious cases, and always when the temperature is high. Uremia has occasionally been noted. After erysipelas of the scalp there is alopecia, and seborrhœa sicca may occur, which gives rise to permanent baldness. Abscess occurs, and lymphangitis has been noted. Arthritis may result from the extension from the skin to the joint tissues. Peritonitis and malignant endocarditis have been seen as sequels.

Diagnosis.—Depends upon the occurrence of the eruption with well-defined margin, showing tendency to spread, occurrence of fever, and constitutional symptoms.

Prognosis.—In simple uncomplicated cases occurring in those in previous good health, prognosis is favorable.

Prognosis should be regarded as serious when erysipelas occurs as a complication of any other malady or from surgical accidents or in the puerperal state. It is always serious in cachectics and in alcoholics.

Treatment.—Prophylaxis is that of the infectious diseases in general. In hospitals erysipelas is isolated and treated in separate wards, as the disease is mildly contagious. Treatment should be directed to the alleviation of the principal symptoms. Free purgation is useless and unsafe, but gentle laxatives at the onset are of advantage.

Water, especially cold water, should be liberally administered to the patient, and cold spongings, especially if the temperature is high, are of distinct advantage.

For the eruption the best treatment consists of iced cloths, frequently renewed, kept over the eruption. An ointment of ichthyol and lanolin is also used for this purpose, but this is smeary and the benefit derived is questionable. Collodion may be painted over the eruption with good result. There is no specific treatment.

Tincture of chlorid of iron in full doses is the general method of treating erysipelas. It is, however, questionable whether any good has been obtained from its use. In severe

cases the hypodermic use of pilocarpin, as first advised by Da Costa, is of distinct benefit, if cautiously used. The physiologic effect of pilocarpin should be obtained, but a stimulant should be administered at the same time, on account of the depressing effects of the pilocarpin on the circulation. When nervous symptoms become prominent, or in the aged or cachectic, bold stimulation is necessary. Alcohol is best for this purpose. If the pain be severe, the hypodermic injection of morphin should be resorted to. Systematic and liberal administration of nourishment must be insisted upon in severe cases. Antistreptococcic serum may be beneficial, and may be resorted to, especially in malignant cases.

FEVERS WITH MARKED LOCAL MANIFESTATIONS.

CROUPOUS PNEUMONIA.

Definition.—An acute, infectious, febrile disease, with a characteristic local pulmonary lesion and marked constitutional symptoms.

Synonyms. — Lung fever; lobar pneumonia; fibrinous pneumonia; pleuropneumonia; pneumonitis.

Etiology.—Predisposing Cause.—Climate is a predisposing cause, pneumonia being more prevalent in warmer than in colder climates. It may occur at any season of the year, but especially in the winter and early spring. Nine-tenths of the cases of pneumonia of the aged occur between November and May. Sudden changes in the temperature have greater influence on the production of pneumonia than prolonged steady cold weather. Damp weather and rainy seasons do not predispose.

It occurs at all ages, and is the most fatal of all diseases after sixty. The male sex suffers to a greater extent than the female, probably due to exposure and occupation. Depressing influences, both physical and mental, are said to be predisposing causes. Previous disease is important, as pneumonia is often the terminal event in the cachectic and alcoholic individual. It is a common sequel in acute diseases, such as the malarial and other infectious fevers.

Exciting Cause.—The specific organism is the diplococcus pneumoniæ. (For a description see p. 103.) Other organisms

have been found associated with croupous pneumonia, such as Klebs-Löffler bacillus, staphylococcus, bacillus typhosus, bacillus coli communis, and bacillus of influenza. It is only within the last decade that pneumonia has been widely recognized as an infectious disease, up to that time having been regarded generally as a local inflammation of the lungs.

Period of Incubation.—Period of incubation is unknown.

Pathology.—This form of inflammation of the lungs affects most generally a lobe, or more than a lobe, sometimes affecting the entire lung; hence the name, *lobar pneumonia.* The most frequent site of attack is, first, the right base; next, the left base; both bases and the apices less frequently. The condition is a process of acute inflammation, the stages gradually merging one into the other, and, for the purpose of clearness in description, the stages are defined separately.

The pathology of croupous pneumonia is divided into three stages: first, *engorgement;* second, *consolidation;* third, *resolution.* The infectious irritant lodges in the lung, and produces its effects in the air vesicles. The introduction of the infection is imported into the lung through the respiratory tract. The diplococcus of pneumonia being in many instances normal in the saliva, the mode of infection is, therefore, easily traced when the system is in such a condition as to favor the growth of this organism.

In the stage of congestion the blood-vessels are found dilated, the lung becomes red, and when the affected area is placed in water, it floats at a deeper level than normal tissue. Microscopically, it will be found that in the stage of engorgement the blood-vessels are dilated, but little or no exudate is noticed in the air vesicles.

The epithelium lining the air vesicles becomes granular, swells, and is shed of its basement membrane. Gradually, the exudate forms in the air vesicles, giving rise to the stage of consolidation, the first part of which is termed *red hepatization,* so called because it resembles liver structure. In this stage the affected area is completely consolidated, and when placed in water, it sinks. There will be complete absence of crepitation. Microscopically, it will be found that the exudate in the air vesicles is composed of a large amount of fibrin, leukocytes, red blood-cells, and a few epithelial cells. The specific microorganism is found in this exudate. The blood-vessels are still dilated and tortuous. This exudate, as a rule, contracts somewhat, and probably by the force of gravitation seeks the de-

pendent portion, so that it will appear free from the walls of the alveoli except at the lowermost portion. The large amount of fibrin present has given to this condition the name of *fibrinous pneumonia.*

The inflammatory exudate soon undergoes fatty degeneration. The leukocytes increase in number, and this change gives the consolidated area a gray color, called *gray hepatization.*

The stage of consolidation gradually merges into the *stage of resolution,* this being brought about by further degeneration and softening of the exudate, a large part of which is carried away by lymphatics and the blood, some portion being expectorated. The epithelial cells are again reproduced from the bronchial epithelium, gradually extending into the alveoli. If recovery occurs, the lung entirely returns to its normal condition, making it impossible to say whether it has ever been affected by this acute inflammatory process.

The stage of resolution may be somewhat delayed. This is known clinically as "delayed resolution." Instead of resolution, abscess formations may rarely occur, or, in extreme cases, entire death of the infected part, producing gangrene. The inflammatory process may become more or less chronic, fibrid pneumonia resulting. Tubercular infection may arise during or after this inflammatory process.

If the irritant reaches the pleura, which it does in the majority of cases, an acute fibrinous inflammation is set up in this serous membrane, the changes being similar to those just described; hence the synonym, *pleuropneumonia.* The bronchial tubes show a slight amount of inflammatory change. This is probably secondary to the alveolar inflammation. The heart muscle shows cloudy swelling, with hypertrophy and dilatation of the right ventricle and auricle. From extension of the inflammatory process the pericardium may be acutely inflamed. Other internal organs may also evince evidences of cloudy swelling.

Symptoms.—The symptomatology varies in individual cases, presenting marked differences in the onset and course of the disease. This has led writers to divide the cases into the "sthenic" and "asthenic" varieties. On the one hand, the symptoms may point to marked pulmonary disturbance, whereas on the other hand they may be obscure, and the diagnosis can be made with difficulty and only after a careful physical examination. The disease usually begins with a

marked, well-defined, severe chill, which may come on at any time of the day, but more often at night. In scarcely any other acute infectious disease is the chill so prolonged and so severe as in croupous pneumonia.

In other types of the disease, especially in pneumonia of the aged and in alcoholics, the chill may not be present at all. Following it there is an abrupt rise in the temperature, ranging from 103° F. to 105° F., attaining its maximum early—often at the end of twenty-four hours or early on the second day. Following this there is pain, referred to the region of the nipple upon the affected side. The pain is due to the involvement of the pleura, which takes place in the majority of the cases.

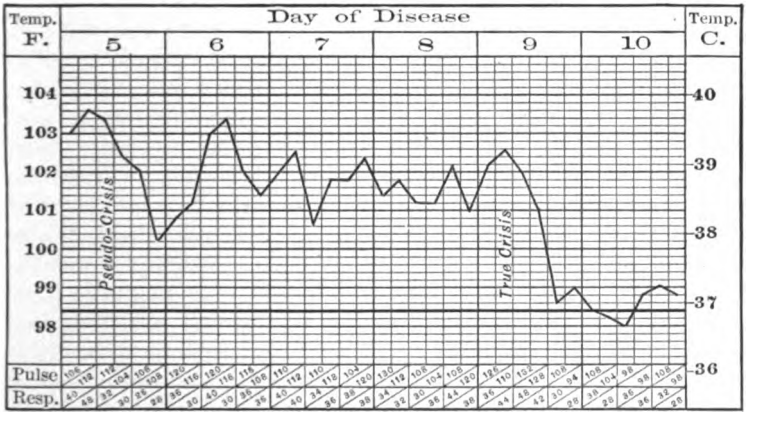


FIG. 31.—Temperature-curve of croupous pneumonia. J. L., twenty-seven years old (Philadelphia Hospital), March 19, 1900.

If the inflammatory conditions do not reach the periphery of the lung, as in central pneumonia, the pleura is not affected and the pain does not occur. Coincident with the pain there is marked dyspnea. The appearance of the face is characteristic: there is an anxious look, a flush upon one or both cheeks, the eyes are bright, and the alæ of the nose deviate with each respiratory act.

Herpes may be present at the nose or at the lips. Cough is now present, which at first is suppressed on account of the severe pleural pain, the patient frequently lying upon the affected side. The sputum early is scanty and viscid, and may be rusty. If rusty sputum does not occur early, it will prob-

ably appear later in the course of the disease, especially in well-marked cases.

Nervous symptoms are common—headache, restlessness, and in some cases delirium, although these are not constant symptoms. Backache, anorexia with great thirst, constipation, scanty and febrile urine, also occur. This stage usually lasts about four days, resolution occurring between the fifth and the eighth day; occasionally earlier and sometimes later.

In typical cases the temperature falls abruptly by crisis, usually upon one of the odd days of the disease. In other cases lysis occurs. During the entire course of the disease there are well-marked physical signs, which may continue for several weeks, even after resolution has taken place.

Description of Special Symptoms.—*Temperature.*—The temperature rises abruptly to 103° F. or 105° F. and even higher, attaining its maximum early. The morning remissions and evening exacerbations amount to about one degree, the temperature in ordinary cases running a subcontinuous course. Occasionally remissions occur, the temperature falling from two to four degrees, but not reaching the normal. These are known as pseudocrises and may precede the true crisis. The true crisis may be preceded by a rise in the temperature, known as the precritical rise. The crisis occurs usually upon the fifth, seventh, or ninth day of the disease, and may take place at night, the temperature reaching the normal or subnormal ranges. Copious sweating takes place at this time, or there may be profuse diarrhea, and large quantities of urine may be voided.

Defervescence occasionally comes on by lysis, especially in the asthenic types. An elevation of temperature occurring after defervescence is due to complications. In pneumonia affecting an apex hyperpyrexia is very apt to occur.

Respiration.—The respirations may number from thirty to forty a minute: they are more frequent in children and in nervous patients. The ratio of respiration to pulse, which in the normal condition is one to four, may be from two to four, or even one to one. The breathing is painful, shallow, and partly suppressed, especially if the pleura be affected; the pain lasts several days from the onset of the disease. Cough may be frequent and distressing, but in the asthenic cases, in the aged, and in alcoholics, as well as in central pneumonia, it may be entirely absent. It is unproductive, short, and dry at first, sputum showing itself in a day or two.

Sputum.—The sputum varies according to the stage of the disease. It is usually moderate in quantity, but may be absent altogether. It is at first glairy, mucous, viscid, and frothy, and may be stained with blood. In the stage of hepatization it becomes gelatinous and very tenacious, so that if it be expectorated into a cup and this be inverted, the sputum will cling to the sides of the vessel and not fall. It shows various shades of red, small streaks of blood being scattered through it, looking like iron rust; hence the name, "rusty sputum." This is a characteristic phenomenon of this disease. In severe cases in which the hemorrhagic element is profuse, especially in alcoholics, the sputum is more fluid, having the character of prune-juice, and is therefore called "prune-juice" expectoration. Microscopically, it contains white and red blood-corpuscles, degenerated and pigmented alveolar epithelium, and bacteria, in which the pneumococcus is usually found to be present. The sputum may contain fibrinous casts of the bronchi, and in the stage of resolution is thick, of a yellow color, and of a mucopurulent character.

Pulse.—The pulse is related to the height of the fever, being from 120 to 130, and in children and asthenic cases it is even more rapid than this. The pulse-rate falls with crisis, and may be as low as 50 a minute. Early in the disease the pulse is full and strong, but later it becomes compressible and irregular.

Nervous Symptoms.—Nervous symptoms are present in the old, the very young, and in drunkards. Headache is common, and there may be a mild delirium at night or the mind may remain clear during the entire attack. In cases with high temperature in which the apex is affected, delirium is a prominent symptom. In children convulsions may occur, the clinical picture closely simulating meningitis. In drunkards delirium tremens commonly takes place.

Digestive System.—Anorexia is complete, and there is much thirst. Vomiting is commonly present in children; constipation is the rule, but diarrhea may be present from the onset. If it appear late in the disease, it is an unfavorable symptom.

Urine.—The urine shows the characteristics common to the fevers. It is scanty, of a high specific gravity, and in one-third of the cases contains albumin. During the course of the disease the urates are generally increased, but the chlorids are diminished. This condition is considered diagnostic by some authorities.

Blood.—The blood shows the presence of leukocytosis. This may be as high as 20,000 to 50,000 in a cubic millimeter.

The patient commonly lies upon his back or upon the affected side, the nostrils expanding with each inspiration and the cheeks are flushed on one or both sides.

Jaundice, if present, and occurring early, may be due to catarrh of the gastro-intestinal tract or congestion of the liver; if late, it is hemahepatogenous in origin. Febrile jaundice, according to Leube, is always suspicious of croupous pneumonia. Herpes of the nose and lips is common, occurring in at least one-third of the cases, and usually early. Sudamina show themselves when sweating appears.

Physical Examination. — Inspection.—Upon inspection the respiratory action of the affected side is restricted, usually without bulging. If bulging occur, it is most probably due to an intercurrent pleurisy with effusion.

Palpation.—Upon palpation over the affected area there is an increase of vocal fremitus. This may at times be absent, if the main bronchus be occluded by tough masses of the exudate or if pleural effusion be present.

Percussion.—At the beginning of the attack percussion over the area of the lesion is clear, since the process of exudation has yet scarcely begun. When exudation takes place, impairment of the resonance occurs, which may go on to complete dullness. If pleural effusion be present, there may be flatness. Above the area of consolidation "Skodaic resonance" is found upon percussion. As resolution occurs the dullness gradually gives place to impairment of resonance, and finally normal pulmonary resonance is again encountered.

Auscultation.—These phenomena depend, first, upon the presence of an exudate that is yet fluid, and, second, upon the presence of an exudate that has undergone coagulation, and thus converted a portion of the lung or the entire lung into a dense, practically airless tissue. At the beginning of the disease there are large and small bronchial rales that are not to be distinguished from those occurring in bronchitis, which is indeed an associated condition. In addition to this, in most instances early in the attack, the crepitant rale occurs, which is characteristic of croupous pneumonia. In the stage of consolidation the crepitant rale disappears as the vesicular structure of the lung becomes entirely filled with the exudate. The rale reappears in the stage of resorption (third stage). (For

description of Crepitant Rale, see p. 74.) Increased vocal resonance is present in the stage of consolidation.

The breathing, which at first is bronchovesicular in character (harsh respiration), later becomes bronchial in the stage of consolidation, and as absorption takes place the crepitant rale and a mixture of bronchial rales reappear, and are known as the "crepitus redux." Associated physical signs upon auscultation relate to the pleura. Friction sounds, more or less distinctly audible, may be present with friction fremitus.

When pleural effusion occurs, the physical signs relating to this condition are present. If pleural effusion be suspected, exploratory puncture should be made as early as possible.

Varieties.—Pneumonia in Children.—Croupous pneumonia is by no means a rare affection in childhood. The initial chill is usually absent. Vomiting is more common than in the adult. Nervous symptoms may be marked from the onset, such as drowsiness, delirium, and convulsions, which may completely mask the physical signs, as rusty sputum is rare in children, and, if it occurs, is usually swallowed. The temperature in childhood is high. The disease commonly affects the apex.

Pneumonia in the Aged.—This may begin as a typical pneumonia, but as a rule the onset is more gradual. Prodromes may be present; rusty sputum is rare, and the temperature is not so high.

Pneumonia in Alcoholics.—Pneumonia is of common occurrence after a debauch or in individuals subject to chronic alcoholism. Delirium tremens may appear early, and the accompanying nervous symptoms completely mask the other phenomena of the disease. There is no cough, pain, nor shortness of breath. The temperature may be but slightly elevated or even subnormal, and it is only upon careful physical examination of the chest that a diagnosis can be made.

Pneumonia with Slight or Central Pulmonary Lesions.—The onset and subjective symptoms are definite and well marked, but the physical signs may be few or absent altogether. If the lesion occur in the interior of the lung and does not reach the periphery, the typical symptoms may be present, but the physical signs may be entirely wanting. The examination of the sputum in such cases may be important. This condition is known as **central pneumonia.**

Typhoid Pneumonia.—This name is misapplied by practitioners to two varieties of cases: First, pneumonia of the asthenic variety, occurring with the so-called "typhoid"

state; second, to pneumonia occurring as a complication in enteric fever. The term *typhoid pneumonia* had better not be used, as it is misleading and not at all distinctive.

Pneumonia Occurring as an Intercurrent Affection in Chronic Diseases.—This is common in nephritis, diabetes, locomotor ataxia, and other diseases of the spinal cord. It may also complicate tuberculosis and chronic bronchitis. Occurring in the course of chronic diseases, it retains its characteristic phenomena and should be regarded as an independent affection.

Complications.—Complications in pneumonia are few. Bronchitis and pleurisy should be regarded as accompanying the disease. Pleurisy with effusion and empyema occasionally complicate this disease. Pericarditis occurs as a complication, especially in left-sided pneumonia, particularly from extension due to the disease of the pleura. Endocarditis, especially malignant endocarditis, may be a complication. Peripheral neuritis is rare, as is also parotid bubo. Hemorrhages occasionally manifest themselves in the alcoholic variety.

Diagnosis.—This rests upon the suddenness of the onset, with a characteristic chill, high temperature, nature of the sputum, pain, herpes, and the defervescence by crisis.

Differential Diagnosis.—This disease must be differentiated from pleural effusions and bronchopneumonia.

Croupous Pneumonia.	*Pleural Effusion.*
Sudden onset, with chill and high fever.	Onset moderate; no chill; slight fever.
Dullness on percussion. If it occur at upper part of the lung the lower portions are clear.	Flatness upon percussion always at the base extending upward.
Vocal fremitus and vocal resonance increased.	Vocal resonance and vocal fremitus absent or diminished.
Bronchial breathing.	Absent breath-sounds.
Crepitant rales, before and after stage of dullness.	Friction sound may be present early in the disease, disappearing when effusion takes place.
No displacements of organs.	Marked displacement of organs—heart, liver, Traube's semilunar space.
Rusty sputum.	Sputum, if present, *never* rusty.
Crisis upon fifth, seventh, or ninth day, with critical discharges.	Lysis, no critical discharges, and duration of the disease more chronic.

Croupous Pneumonia.	*Bronchopneumonia.*
Primary disease, with sudden onset.	Always secondary to bronchitis.
Often unilateral.	Always bilateral.
Affecting young, robust adults.	Occurs at the extremes of age, and never affects young adults.
Typical temperature.	No typical fever-curve.
Rusty sputum.	Sputum as in bronchitis, never rusty.
Crepitant rales and crepitus redux.	Subcrepitant rales.
Duration brief, ending by crisis.	Duration prolonged, ending by lysis.

Prognosis.—In the robust type the prognosis is favorable, most cases ending in recovery. At the extremes of life and in alcoholics the prognosis is grave. When pleurisy with effusion occurs, the prognosis is more unfavorable. Endocarditis and pericarditis increase the gravity of the case. An intense toxemia is very serious. When a number of cases appear in the same house or in a restricted locality in which unhygienic surroundings prevail, the prognosis is unfavorable. The mortality from croupous pneumonia is generally estimated at about 20%.

Treatment.—There is no prophylaxis, as the period of incubation is unknown. One attack is not protective: on the contrary, it rather predisposes to other attacks. In young adults, with sudden engorgement of the right heart and signs of pulmonary congestion and edema, with full, hard pulse, blood-letting is not only permissible, but even necessary to save life. In the old and in the alcoholic variety this is a dangerous procedure.

A room with good ventilation should be selected. The food should be simple and of the ordinary variety given in fevers. It should be sparingly given early in the attack, and freely during convalescence. A laxative medicine at the onset is of use. The fever usually does not require treatment, as the disease is a short one. Cold sponging may be practised morning and evening. If hyperpyrexia occur, the cold bath may be resorted to. The modern antipyretic analgesics must be used with caution. For the pain, several ice-bags should be applied to the affected side, or moderate doses of opium in some form given, such as Dover's powder in doses of from three to five grains, which may be administered every three or four hours until the pain is relieved or light somnolence occurs.

Poultices and the cotton jacket are of exceedingly doubtful utility, and prevent the necessary and systematic examinations of the chest. Alcohol may be given in small doses from the beginning of the attack, to control the nervous symptoms, and especially in the asthenic forms. Strychnin as a cardiac and respiratory stimulant is useful, and may be given either by the mouth or hypodermically. For sleeplessness, especially in the alcoholic variety, chloral should not be used. Trional and sulphonal are safer drugs.

If there be evidences of contraction of the peripheral circulation, the nitrites, especially nitroglycerin, are useful. Oxygen

inhalations should be administered in all severe cases. Blisters should not be applied at the height of the disease, and should only be resorted to in cases of delayed resolution.

Convalescence is usually rapid, tonic treatment being, therefore, unnecessary. Complications must be treated as in any other affection, and upon their own merits.

ACUTE RHEUMATIC FEVER.

Definition.—An acute febrile disease, characterized by inflammation of the joints, acid sweats, and a tendency to involvement of the serous membranes, particularly of the joints and the heart, with constitutional symptoms.

Synonyms.—Acute inflammatory rheumatism; rheumatic fever.

Etiology.—The disease shows a hereditary tendency, and in the new-born and in young children it is more likely to occur in the female, in the proportion of five to one. In later life the male sex is more liable, probably due to increased exposure. Age is an important predisposing factor, the disease occurring particularly in young adults; it may, however, arise at any age. It is more prevalent in cold and damp climates, although it is not infrequent in the tropics. It is an unknown disease in some parts of Belgium.

Exposure to cold and injuries to the joints are not particularly predisposing causes. On the other hand, changes in the weather and prolonged exposure to such changes are important factors. Acute rheumatic fever attacks all classes, the well-to-do and the poor suffering equally.

Occupations such as those of coachmen, drivers, scrubbing women, etc., that require continual long exposure to cold and wet, predispose.

The specific cause of acute rheumatic fever is by no means settled. By some it is still regarded as a mere inflammation of the joints. However, the view that the disease is an infectious one is constantly gaining ground. Three principal theories have been entertained as to the causation of this disease. These are:

1. **The Chemic Theory.**—It is held that acute rheumatic fever may be due to the excess of lactic or uric acids in the tissues. This theory has now been abandoned.

2. **The Nervous Theory.**—This was first advocated by J. K. Mitchell, on account of the fact that joint implication

occurs in a great many of the diseases of the nervous system, such as myelitis, locomotor ataxia (Charcot's joints), chorea, etc.

3. The Infectious Theory.—This was advocated by Heuter, and numbers of modern clinicians are following his views. Various germs have been found from time to time in the blood-serum and in the synovial membranes, but no constant variety has been found uniformly present.

The following reasons make it extremely likely that acute rheumatic fever is an infectious disease: The disease occasionally occurs in an epidemic form; it is self-limited; it mainly affects the young; severe symptoms and complications occur as in other infectious diseases, such as hyperpyrexia, endocarditis and pericarditis, pleurisy, and pneumonia; there is a marked tendency to leukocytosis, albuminuria, and anemia; rashes not infrequently occur; many infectious diseases also combine joint affections, such as pyemia, scarlatina, cerebro-spinal fever, etc.; the toxemia is best explained by the variety of different symptoms, and relapses occasionally take place.

Pathology.—There are no constant lesions found after death. Most patients recover without permanent lesions in the joints, and unless cardiac complications follow, are wholly free from pathologic remains. The joints during the acute stage show some degree of hyperemia, especially pronounced in the synovial membranes. There is uniform swelling of the joint structures and ligamentous attachments from inflammatory changes. The synovial fluid may be increased, becoming turbid from flakes of fibrin and leukocytes. Pus and blood in the joints are exceedingly rare.

The blood shows important changes. In perhaps no other infectious disease, with the exception of diphtheria, pyemia, and septicemia, is the anemia so marked and so early produced. The red blood-cells may be reduced one-half or more in number, the hemoglobin reduced to 50%, and leukocytosis is common. The serous membranes of the heart are involved in about one-third of the cases, the left side being most often affected. Ulceration of the valve structure is rare. Myocarditis is not uncommon.

Period of Incubation.—The period of incubation is unknown.

Symptoms.—The disease begins suddenly, with fever rising to 102° or 103° F., with pain, tenderness, swelling, and redness in one or more of the joints. Most often the large joints

are affected. Occasionally, prodromes are present. They consist of headache, lassitude, coated tongue, anorexia, constipation, and chilliness, with mild tonsillitis, pharyngitis, or laryngitis. Epistaxis may be present. These prodromes may precede the sudden onset by two or three days. The joint involvement is quite characteristic in the fact that the inflammation shows a tendency to involve the joints symmetrically: that is to say, if the left knee is affected, the next joint likely to become involved is the right knee, etc. These joints are exceedingly painful, tender to the touch, red, and swollen.

The pulse is accelerated from 100 to 105 a minute, soft and compressible. The temperature varies between 102° F. and 104° F. It shows no characteristic type, and rises with the occurrence of complications. Hyperpyrexia occasionally takes place, with marked symptoms of headache and delirium, which later may be violent in character and end in coma. This has been called "*cerebral rheumatism*," and is extremely fatal. The urine is highly colored, of high specific gravity,—1025 to to 1040,—scanty in amount, contains an abundance of phosphates or urates and occasionally slight amounts of albumin (toxic). The saliva is strongly acid. Nervous symptoms, as a rule, do not appear, as the mind is clear throughout, except when hyperpyrexia occurs, with symptoms that have already been described. Copious acid sweats are characteristic of this disease.

Urticaria and petechiæ occasionally occur, as do erythema nodosum or erythema multiforme. Nodules varying in size from a pinhead to ⅓ of an inch in diameter appear; these may be extremely numerous, painful, and tender, occurring in children particularly, and lasting several weeks and then disappearing.

Follicular tonsillitis has been so frequently observed that by many a causative relation is supposed to exist between these two affections.

One attack does not confer immunity, but, on the contrary, rather predisposes to other attacks.

Complications. — Endocarditis and pericarditis occur in about one-third of the cases. By many these are considered as symptoms, and are not classed as complications. Pleurisy also is not infrequent, as is involvement of other serous membranes. Tonsillitis, bronchitis, bronchopneumonia, and chorea are noted as complications.

Diagnosis.—This depends upon the abrupt onset, often with

a preceding tonsillitis or pharyngitis, with an inflammation of a number of joints, usually the large joints, with fever, profuse sweating, and a marked tendency to implications of the serous membranes.

Differential Diagnosis.—Differential diagnosis must often be made between rheumatic fever and pyemia, and this is usually a difficult matter, particularly if they occur in the lying-in woman. In rheumatic fever a very important point is that the joint affection is usually of a fugitive character, coming and going with great rapidity, whereas in pyemia the joint involved is apt to persist throughout the process. Pus is present in the affected joint in pyemia; this is extremely rare in acute rheumatic fever.

Differential diagnosis between gout and rheumatic fever may be made from the fact that gout usually comes on very suddenly, and shows a special preference for the smaller joints, most usually the great toe. The appearance of the joint is swollen and glossy, tense and bluish. In gout there is less sweating and less fever; gastric and nervous symptoms are more likely to be present.

Course and Duration.—Prognosis.—As a rule, this is a benign disease, terminating in recovery. It may, however, result fatally, from the intensity of the febrile process or from endocarditis or general hemorrhagic tendencies. In mild cases the process may come to an end in about a week; in others the disease may last for months. The severity of the disease usually corresponds to the number of joints affected, but bears no relation to the cardiac complications, as severe endocarditis may result from but a slight joint implication. Recovery takes place even from cardiac complications, but complete recovery is rare, the acute forms of cardiac disease usually passing into the chronic variety. Cerebral rheumatism is almost invariably fatal.

Treatment.—Rheumatic fever is fortunately one of those diseases for which it may be almost claimed that we possess a specific in salicylic acid and the salicylates. These drugs should be given in sufficient quantity to produce their physiologic effect. The drugs usually selected are either salicylic acid—which should never be given in form of a powder, but in capsules—or preferably the salicylate of sodium. An effort should be made to produce the physiologic effect of the drug in from thirty-six to forty-eight hours. Under treatment of this kind swelling subsides, redness disappears, and the tem-

perature falls rapidly. The treatment should be continued for some time after the symptoms of the disease have entirely ceased. If heart affections occur, especially early, it may be good practice to give some of the alkalies or an alkaline and salicylate treatment from the onset. This must be continued until the urine becomes distinctly alkaline in reaction. If the suffering of the patient is great, some form of opium should be administered.

A light, easily digested diet should be used. Light farinaceous foods, rice puddings, broth, and custards are preferable, but concentrated beef-broths and acid fruits should be prohibited.

In convalescence some attention should be given to the high grade of anemia that almost invariably follows acute attacks. The local treatment for the joints should consist in their being wrapped in carded wool. Hot applications with ichthyol to the joints are useful.

Rest is of great importance even in the mildest cases, as frequently the cases that show least joint affection may be the ones to show serious cardiac affection. The rheumatic hyperpyrexia must be treated by ice to the head, or a tepid bath gradually cooled. Bold stimulation is necessary in a condition of this kind.

Treatment of the Cardiac Complications.—When cardiac complications develop, the general idea is to change from the salicylates to the alkalies, such as sodium bicarbonate, potassium citrate, or potassium acetate, or some combination of these.

Large blisters over the heart are of no use, as they add to the suffering of the patient and prevent systematic examinations. Small blisters at some distance from the heart, on the other hand, are often beneficial. Rest should be absolute for at least two or three weeks after the joint affection has subsided, as the heart beats eight to ten times less per minute in the recumbent posture, and this induces necessary heart-rest. Digitalis should be used only for special indications.

DIPHTHERIA.

Definition.—Diphtheria is an acute specific contagious disease, due to the Klebs-Löffler bacillus, characterized by the formation of a false membrane, principally upon the mucous surfaces and occasionally upon the skin, with fever and marked constitutional symptoms. The disease is not self-protective.

Synonyms.—Putrid sore throat; membranous croup.

Etiology.—It is a disease of all climates and seasons, occurring especially in densely populated centers, but when introduced into rural districts it prevails with great virulence. Social condition is without influence as a predisposing cause. Filthy surroundings and bad drainage are favorable soils for its development.

Age is an important predisposing factor, it being particularly a disease of childhood; no age, however, is exempt. The sexes are alike liable. All catarrhal conditions of the mucous membranes of the throat and nose predispose. One attack does not confer immunity.

Exciting Cause.—The Klebs-Löffler bacillus is the exciting cause. (For description of germ, see p. 111.)

Pathology.—The false membrane is usually confined to the pharynx, but may involve the inner surface of the cheeks, lips, skin, tonsils, pillars of the fauces, uvula, soft palate, posterior and anterior nares, Eustachian tube, and the middle ear. In rare instances the trachea, bronchi, esophagus, and stomach are covered with membrane.

The amount of membrane varies from a small patch (frequently situated on the tonsil) to an extensive exudate, which may block up the pharynx, larynx, or nares.

The pseudomembrane is of a dirty-gray color, the mucous surface surrounding it being red, swollen, and edematous. The membrane may be readily stripped off, and leaves a swollen injected surface beneath.

Microscopically, the free surface of the exudate is covered with diphtheria bacilli, and perhaps groups of cocci or other bacteria. The membrane consists of fibrin, numerous small round cells, red blood-cells, and leukocytes, many of which are disintegrated.

When the exudate has gained a firm hold, the epithelium will be found partially or completely absent, the mucosa being continuous with the false membrane. The submucosa is infiltrated with round cells, leukocytes, and red blood-cells. The lymphatics and blood-vessels are greatly dilated. In the inflammatory areas diphtheria bacilli are found. The separation of the membrane in many instances is due to the outpouring of secretion from the mucous glands.

Pneumonia, endocarditis, acute nephritis, and neuritis may occur as complications.

Period of Incubation.—From two to five days.

Symptoms.—The onset of the disease is rather rapid than abrupt. The early symptoms are discomfort and weakness, with headache and general malaise. Moderate fever is usually present at the onset. Pain in swallowing is often the earliest symptom that draws attention to the case. This may not be pronounced, and in very young children is not complained of. It should, however, be the rule of the physician always to examine the throat when he is called to see a child who is taken sick with slight constitutional disturbances. Redness of the soft palate, with more or less swelling of the tonsils, are common. Upon one or both of them, upon the half arches of the palate, or, less frequently, upon the pharynx or hard palate small spots that are grayish-white in appearance show themselves. These spots, which are the first signs of the false membrane, are somewhat thickened and raised above the level of the surrounding mucous membrane, which is deeply congested, especially around its borders. If an attempt be made to forcibly detach these points, a bleeding surface is left beneath, and the membranes quickly reform.

The exudate shows a tendency to spread, and for the first few days this extension may be rapid and extensive, involving all the surfaces. The toxemia is proportionate to the extent of the exudate. In severe cases the entire throat may be covered in a few hours. The extension may be upward toward the nasopharynx and forward into the nasal chambers, into the Eustachian tubes, or more often downward into the larynx, through the glottis into the bronchial tubes. The involved area varies greatly. Usually, it is limited to one or both tonsils, extending a little upon the corresponding half arches of the palate or the edge of the uvula.

The most common position for the exudate is upon the tonsil, less commonly upon the side of the uvula of the affected tonsil. There is early noticeable swelling of the lymphatic glands at the angle of the jaw. If the tonsils are not involved and only the larynx implicated, the lymph-glands in this locality are not found enlarged.

The constitutional symptoms become intensified as the exudate spreads. There is anorexia, thirst, deglutition is painful and difficult, and vomiting frequently takes place. The tongue is moist and coated with a thick yellowish fur. The fever is of irregular course, often rising to 104° F., though ordinarily it is of moderate intensity, and even in grave cases there may be low temperature, which is due to shock. The high temperature

subsides in from three to four days. A continued high fever must be ascribed to some complication.

The mind, as a rule, is clear, delirium and convulsions are rare. The pulse is rapid and weak. In mild cases the constitutional symptoms ameliorate in from eight to ten days, and the membrane disappears with moderate rapidity. Albuminuria occurs early—upon the second or third day. In grave cases the disease may terminate fatally with great rapidity. If the membrane extends into the nasopharynx and nasal chambers, the toxemia is intense, and the symptoms are not necessarily due to faulty nasal respiration. The lymph-channels in the nasal chambers are extensive and relatively large, so that toxic material is readily and quickly absorbed.

In nasal diphtheria there may be a mucopurulent discharge streaked with blood at the nose, and epistaxis is common.

Laryngeal Diphtheria.—Diphtheria of the larynx begins with symptoms of laryngitis. There is mechanical interference with respiration, and marked signs of suffocation may take place.

The larynx in rare instances may be affected primarily, the disease spreading to the pharynx later. In other cases the larynx may be affected alone, but it is more usual for the membrane to spread from the pharynx to the larynx.

The *symptoms of laryngeal diphtheria* are hoarseness, croupy cough, labored and difficult respiration, pallor of the face with cyanosis, and paralysis of the laryngeal muscles. Drowsiness sets in, and the patient may die in a comatose condition. Occasionally, in coughing the false membrane is dislodged, and recovery may take place if the membrane does not reform. This is, however, a rare occurrence. Tubular casts of the smaller bronchi may be coughed up. From extension the membrane may reach the bronchi and alveoli of the lung, and produce bronchopneumonia. It may be due to this bronchopneumonia that relief in some cases of laryngeal diphtheria does not follow tracheotomy. The toxemia alone is capable of producing a fatal issue. In such cases the pulse becomes weak and rapid, and cardiac asthenia will be pronounced. This may occur without stenosis of the larynx.

Septicemia from mixed infection may take place. Heart failure may develop, and prove rapidly fatal. This may occur during convalescence. Often between the second and third day the urine contains albumin, casts, and traces of blood. Edema, when present, is usually slight.

Paralysis.—Diphtheric paralysis may show itself during convalescence, occurring in mild as well as severe cases. The soft palate is most frequently implicated. Swallowing is interfered with, and fluids regurgitate through the nose in consequence of the inability of the palate to approximate itself to the posterior wall of the pharynx, so that the pharyngeal muscles contract on the substance or some portion of it and force it up into the nostrils. This is a form of motor paralysis. There may also be sensory paralysis. Paralysis of the muscles of the eye occurs; strabismus is common, but paralyses of the trunk and of the extremities are less common. When this condition affects the mechanism of the heart, sudden death from paralysis takes place. The vocal cords may also be affected. The knee-jerks are often abolished.

Diagnosis.—The direct diagnosis is easy. The exudate with the inflamed areola, severe local and constitutional symptoms are quite characteristic. If the tonsils alone are affected, some difficulty in diagnosis may occur.

Follicular or lacunar tonsillitis often simulates this condition, and a bacteriologic test may be necessary. Pseudodiphtheria frequently takes place in scarlet fever, and resembles true diphtheria, and here a bacteriologic examination is necessary to determine the true nature of the affection.

Prognosis.—The death-rate varies in different epidemics, but the prognosis should always be guarded. Favorable conditions are: a limited membrane, slight swelling of the lymphatic glands, and moderate fever. Unfavorable conditions are: marked local inflammation, thick false membrane, great inflammation of the lymphatics, and tendency of the membrane to extend upward or downward. The extension into the larynx renders the prognosis unfavorable, as is also the case in nasal diphtheria.

Treatment.—Prophylaxis.—Prophylaxis is of the utmost importance, the disease being highly contagious and easily transmitted. The patient should be strictly isolated, although the area of contagion is limited. Efficient disinfection is necessary in the sick-room, and at the end of the attack the apartment should be thoroughly disinfected, as should also the bedding, clothing, etc.

The sick-room should have a temperature of between 70° and 75° F. The food must be nutritious and easily digested; milk, animal broths, and predigested foods are useful. If the

patient swallows with difficulty or there is much vomiting, rectal alimentation should be practised. Alcohol is necessary, and must be given freely even in mild cases; however, its influence upon the pulse, heart, and nervous system should be carefully watched.

The membrane must not be removed. Antiseptic and soothing applications are employed; inhalations of quicklime and steam in all cases in which there is danger of invasion into the larynx are useful. Hydrogen dioxid in solution is serviceable as a mouth-wash; likewise, Löffler's toluol solution:

℞. Menthol . . . . . 10 grams.
Toluol . . . . . 36 c.c.
Ferri-sesquichlorid . . . . . 4 c.c.
Alcohol . . . . . 60 c.c. M.

When symptoms of suffocation appear, the patient is placed in a warm bath with cold applications to the head.

Internally, the tincture of chlorid of iron may be given in medium doses. Preparations of mercury, calomel, or corrosive sublimate are beneficial. At the beginning of the attack a laxative dose of calomel is desirable; following this, one-sixth to one-quarter of a grain may be given every two or three hours until free action from the bowels is produced.

The paralysis of diphtheria should be treated with iron, nux vomica, strychnia, and electricity. Rest and a nutritious diet are necessary, and the patient may be benefited by massage. Heart failure and cardiac asthenia require absolute rest and the administration of alcohol; strychnia also is of use in this condition.

The expectant plan of treatment should not be adhered to in a disease like diphtheria. The case must be treated with energy and promptness, and an attempt made to control each symptom as it arises.

Antitoxin Treatment.—The neglect to use the antitoxin treatment in the present state of medical knowledge should be considered almost criminal negligence. The earlier the treatment is instituted, the better the result. The dose in individual cases varies: in severe cases that have lasted some hours or a day or two the injection should be repeated, and from 1500 to 2000 antitoxin units used. From three to four injections may be required. Through this treatment the mortality has been reduced from about 50 per cent. to 20 per cent., or even lower. It should be practised even in sus-

pected cases, and before the bacteriologic examination has been made. In laryngeal diphtheria it may be necessary to resort to intubation or tracheotomy.

Antitoxin Injections.—The skin at the point of selection for the introduction of the serum should be treated as if for a surgical operation, and the syringe, which should have a capacity of from two and one-half to three fluid drams, sterilized. The injection should not be beneath the deep fascia, but in the subcutaneous tissues, preferably in the back between the shoulder blades, or in the outer surface of the thighs, thus preventing the patient from seeing the operation.

The serum should be *slowly* forced in, the needle withdrawn, and the puncture sealed up with rubber adhesive plaster or cotton and collodion, to prevent the fluid from escaping. *The part should not be rubbed.* The dose for immunization, which should always be used when there are small children in the household, is from 200 to 500 antitoxin units, or from 500 to 1000, according to the age of the patient. There is no serious reaction and no danger from the serum itself if pure, therefore full doses may be given.

CEREBROSPINAL FEVER.

Definition.—A malignant continued fever due to the diplococcus intracellularis, occurring in epidemics and occasionally as a sporadic disease, with marked symptoms relating to the cerebrospinal system, and constant anatomic lesions after death.

Synonyms.—Epidemic cerebrospinal meningitis ; epidemic meningitis ; cerebrospinal typhus; petechial fever; spotted fever.

Description.—This disease was first recognized as a substantive affection about 1805. The earliest epidemics occurred in Switzerland and Southern France. The disease appeared in epidemic form in 1863 in the United States, and prevailed annually until about 1870. In 1873 the disease again became epidemic in this vicinity, since which time cases have constantly occurred, sometimes epidemically, occasionally in sporadic form. It is also an epizootic disease, as some of the domestic animals suffer from it, especially the horse.

Epidemics usually run their course in from a few weeks to a few months. They have, however, prevailed for a year or more. The causes of the prevalence of epidemics are not known.

Etiology.—Predisposing Cause.—Climate is of no importance, the affection having occurred equally in warm and cold climates. Season is of some importance, as the majority of the outbreaks have occurred in the spring. Nothing is known in reference to temperature, moisture, or the direction of the wind. Altitude and soil are without influence. Damp, cold, and unclean residing places seem to predispose. Race and social condition are without influence. Among adults a larger proportion of females than males appears to suffer. No period of life, however, is exempt.

Exciting Cause.—The diplococcus intracellularis meningitidis, described by Weichselbaum in 1887. (See p. 102.)

Pathology.—Changes in the membrane of the cord and brain vary from an intense injection of the pia and arachnoid to a profuse fibrinopurulent exudate, the latter being the most common.

An effusion of turbid serum into the subarachnoid space and ventricles is frequently met with.

The exudation is commonly general, the membranes of the base of the brain, posterior portion of the cord, especially in the dorsal and lumbar regions, showing it to a greater degree.

Inflammatory areas of the tissues of the brain and cord may be met with as a result of extensions. The spinal nerve-roots as well as some of the cranial nerves may be covered with the exudate. Microscopically, the exudate is largely composed of fibrin and leukocytes. The diplococcus is found in the exudate.

The spleen is usually not enlarged. Pneumonia, pleurisy, and acute nephritis may accompany the disease. Leukocytosis is present.

Period of Incubation.—This has not yet been definitely settled; it is, however, estimated as being between three and five days.

Symptoms.—The symptoms are related to two conditions —to the toxemia and to the local inflammation. Either of these two groups may predominate, and the symptoms vary accordingly. Prodromes are uncommon; if present, they are not severe, and consist in malaise, slight headache, and stiffness of the limbs. The onset is sudden, beginning with intense *headache*, most frequently at the back of the head. Developing coincidently with this is stiffness in the muscles of the back and the neck.

An initial chill may occur. There is fever with vomiting from the beginning. The vomiting is of the cerebral type. Delirium and stupor may be present early. The intensity of the early symptoms vary in different cases and different epidemics. Violent pains often occur with extreme rapidity, the symptoms appearing with lightning-like quickness. These cases are known as the "*fulminant variety*," in which the patient is overcome by intense toxemia. Nervous symptoms may appear, and the patient succumb in a few hours or days. On the other hand, they may be extremely mild, and the disease end in recovery in a day or two. This is known as the "*abortive variety*." Occasionally in epidemics, cases occur in which the symptoms are so mild and the toxemia so light that they would not be diagnosticated except for the knowledge of an epidemic. These are known as the "*larval*" or "*undeveloped variety*." Severe cases last from two to eight weeks, and may even then terminate fatally.

Description of Symptoms.—The headache is usually occipital, although it may be general; it is always severe. There may be periods of remission, being accompanied by a sense of fullness in the head with vertigo. There are pain in the neck, tenderness along the spine, and the muscles of the spine may contract so that well-developed opisthotonos is present. Delirium occurs early, and the disturbance of the mind may vary from slight drowsiness to profound stupor with wild, maniacal delirium. Convulsions take place in severe cases.

Vomiting is a prominent symptom, and is of the cerebral variety, without much nausea and usually with a clean tongue.

The symptoms referable to the individual nerves are variable, the ones most commonly involved being those of the special senses. Ptosis may be present; nystagmus, strabismus, disturbance of the pupils, and affections of the facial nerve may occur. Trismus may be a symptom. Tinnitus aurium is common. Optic neuritis occasionally shows itself with destructive inflammation of the eyeball and purulent conjunctivitis or keratitis. The sense of smell may be entirely lost (anosmia).

Disturbances of sensation are common, such as hyperesthesia, anesthesia, and paresthesia. Subsultus tendinum is present, and the reflexes may be normal, exaggerated, or absent. The tendon reflex, however, is usually diminished or absent. The fever does not conform to a type, and its severity is not proportionate to the other symptoms. Hyperpyrexia

may occur or the temperature may not be higher than 101° F. Herpes is characteristic and common in cerebrospinal fever, and may be of diagnostic value in doubtful cases. Other eruptions are sometimes present—erythema, urticaria, and petechiæ. Anorexia is present. Constipation is the rule, but diarrhea occasionally takes place. There is no appreciable enlargement of the spleen. Arthritis is occasionally a symptom.

The urine is scanty and has the characteristics of febrile urine. Albumin is present (toxic albuminuria). Polyuria and glycosuria occasionally occur. The respirations are increased in number, irregular, and shallow.

Kernig's Sign.—Kernig observed a flexion contracture of the knee-joint, which in the sitting posture could not, without violence, be straightened beyond an angle of 135 degrees with the thigh, but which was readily straightened when the patient was in the erect or the recumbent posture. Kernig has examined several thousand persons especially for this phenomenon without finding it in a single instance except in cerebrospinal fever.

Complications.—Bronchopneumonia, bronchitis; endocarditis and pericarditis are rare.

Sequels.—Sequels are exceedingly common. They consist in affections of the special senses. There may be loss of sight, permanent deafness, loss of smell, loss of taste, and various forms of paralysis. Gradual recovery may take place from some of these conditions, but this is unusual.

Diagnosis.—In sporadic cases the diagnosis is difficult if the symptoms are not typical. The direct diagnosis rests upon the abrupt onset, grave cerebral symptoms, *associated headache*, *vomiting*, and *painful retractions of the muscles of the neck and herpes* with the presence of Kernig's sign.

If the symptoms of meningitis be present in any case, it should be the duty of the physician to endeavor to ascertain whether it be a primary or secondary affection. It is secondary if it should occur from extension, from disease of the ear, the nose, or to traumatism, from some other infectious process, to brain abscess, and the like. Tuberculosis should always be suspected.

Differential Diagnosis.—The differential diagnosis must be made between cerebrospinal fever and enteric fever.

Enteric fever is more gradual in its onset, the cerebrospinal symptoms occurring at the beginning of the disease. The

fever has a typical curve. Dicrotic pulse is present. Vomiting is not frequent nor pronounced, and ceases early in the course of the disease. The eruption occurs at the end of the first week, and is characteristic. Abdominal symptoms are marked, and the spleen is enlarged early in the course of the disease. Delirium takes place late in the affection. Widal's reaction is important in doubtful cases. Photophobia is rare in enteric fever.

Prognosis.—The prognosis is very grave, the average mortality being 45%. The mortality is higher in the early course of an epidemic. Occasionally, epidemics occur in which all of the symptoms are slight, and very few deaths take place. On the other hand, some epidemics may show principally cases in which fulminant phenomena are prominent.

Treatment.—Prophylaxis is unknown, and the treatment of the individual case is purely symptomatic. Cold applications to the head and to the spinal cord have received the sanction of the best practitioners. On the other hand, applications of heat to the head and spine may be found of use in some cases. A laxative dose of calomel is useful early in the course of the disease, and mercury throughout the entire affection has many advocates. Opium, perhaps, offers the best mode of treatment in this disease, and there is a remarkable tolerance for this drug even in the very young, as enormous doses may be given with benefit. It should be begun in small doses, which should gradually be increased until the effect of the drug is produced. Large doses of opium may be continued for a long period without danger to the patient. If vomiting is a prominent symptom, morphin should be given hypodermically instead of opium by the mouth. Chloral, the bromids, and cannabis indica have been used, but in effect can not be compared with the systematic use of opium. Alcohol should be given according to the requirements of individual cases. It is necessary when depression shows itself and asthenia is marked.

CHOLERA.

Definition.—A specific disease due to the comma bacillus of Koch, prevailing endemically in some parts of the world, occasionally becoming epidemic, characterized by vomiting, purging, muscular cramp, and a high mortality.

Synonyms.—Cholera Asiatica; cholera infectiosa.

Etiology.—The delta of the Ganges is the home of cholera. The disease first made its appearance in Europe in 1830. It occurred in 1835 and 1836 in America for the first time, and from thence outbreaks have occasionally been noted. The disease is not restricted to any climate, epidemics having occurred in Siberia and India. High temperature, however, favors the development of the disease and its spread, but cold weather does not necessarily arrest it. Cholera follows the line of travel, and affects particularly the centers of population, hence it is more likely to appear in sea-coast towns rather than inland cities. Individual immunity does not exist, all persons being susceptible to the disease. All ages are liable, children, however, suffering less in proportion than adults. Neither sex nor occupation have any influence. The mode of life of the individual, unhealthy surroundings, unclean habits of living, and insufficient food increase the danger of infection. Alcoholism, acute and chronic gastro-intestinal catarrh predispose. It is held by competent modern observers that anxiety and fear act as predisposing causes.

Exciting Cause.—The exciting cause is the comma bacillus of Koch, which gains access through the gastro-intestinal tract. (For description of the germ, see p. 116.) The germ multiplies in the intestines, where toxins are thrown off, which are absorbed, and these produce the characteristic symptoms of the disease.

Pathology.—The anatomic changes found in the cadaver are not characteristic. All the signs resulting from extreme collapse are present. There may be postmortem elevation of temperature. The rigor mortis is established early and is very intense, so that changes in the posture of the body may occur; the position of the eyeballs may be changed, the closure of the jaw effected, and the posture of the limbs altered.

The postmortem rigidity persists for some time. The body shows extreme emaciation from loss of fluids. The tissues are dry and doughy, and the outline of the bones may show through the subcutaneous tissues. The blood is dark in color and inspissated, the corpuscular elements are relatively increased. The fluid and salts of the body are diminished. The serous membranes are dry and lusterless. The internal organs show no constant changes. The mucous membrane of the intestines is swollen in many parts, denuded of epithelium, and the specific bacilli are found in its contents. The small intestine contains turbid fluid with epithelium and leu-

kocytes. The spleen is usually small; the liver and kidneys show parenchymatous degeneration. The heart is soft and friable, and the lungs are congested at the bases.

Period of Incubation.—From two to five days.

The Clinical Course of Cholera.

Symptoms.—For the sake of convenience the attack of cholera may be divided into four stages: the stage of *premonitory diarrhea*, the stage of *serous diarrhea*, the stage of *collapse*, and the stage of *reaction*.

Stage of Premonitory Diarrhea.—The patient has the symptoms that are usual in an ordinary acute intestinal catarrh, with colicky pains, followed by large watery stools; there may be from three to eight or ten in the course of twenty-four hours.

There is anorexia, thirst, some headache, and depression with perhaps vomiting. There may even in this stage be symptoms of pain in the calves of the legs and diminished excretion of urine. Slight fever may be present. This stage may terminate in recovery in the course of two or three days, and is sometimes spoken of as "*cholerine*."

Stage of Serous Diarrhea.—On the other hand, in the course of a few hours or a day or two grave symptoms arise. These usually come on at night, and the diarrhea continues, but is no longer fecal but serous. The stools are thin and copious. From absence of bile they lose their normal color and become grayish-white, flocculent in character, looking like rice-water; hence the name, "rice-water stools." They are without odor and alkaline in reaction. Microscopically, they contain epithelial cells, crystals of triple phosphates, the comma bacillus, and other bacteria. The detection of the comma bacillus in this stage renders the diagnosis certain. Serous diarrhea is accompanied by vomiting, which is frequent but is unaccompanied by much retching or distress. The vomited material consists of undigested food and sometimes of the rice-water discharge from reverse of peristalsis. The appetite is lost, thirst is extreme, and the tongue is thickly coated.

This stage lasts from two to seven hours, and may terminate in reaction, but more frequently the patient merges into the stage of collapse.

Stage of Collapse.—The effects of the toxins upon the nervous system are now manifest; the diarrhea is less urgent, although the contents of the intestines may escape continu-

ously and involuntarily. The heart's action becomes more and more feeble. The pulse is almost imperceptible. The respirations are slow, irregular, and shallow. The surface temperature is subnormal,—95° F. or less,—whereas the rectal temperature may show 103° F. to 105° F. The surface is cold; the skin is inelastic, livid, and wrinkled, and shows evidence of profound collapse; it is dry; the fluids are not secreted. If urine is passed at all, it is of high specific gravity and contains albumin. Cramps in the muscles are prominent. There are convulsive movements of the legs, arms, hands, etc., which constantly recur. The mind, as a rule, remains clear. Great restlessness and excitement are exceptional. This stage may last from a few hours to a day, and when death occurs in this stage, it is preceded by coma.

Stage of Reaction.—This may succeed the stage of collapse. Instead of the case ending fatally, the evacuations become less frequent and copious, and cyanosis gradually disappears, the surface becoming warm. Vomiting ceases. Copious perspiration may occur. Secretion of urine increases, but this may contain albumin, casts, and blood-corpuscles. If the progress of the case continue favorable, the urine becomes normal, the internal temperature falls, and the external temperature rises.

This condition may be interrupted by a relapse, and the symptoms previously described may recur; or the stage of reaction may, at the starting-point, develop a secondary infective process. This is known as "*cholera typhoid.*"

Cholera Typhoid.—High temperature occurs, with headache, stupor, and delirium; full, frequent, bounding pulse, with flushed face; an eruption of an erythematous character may appear upon the chest or extremities. Symptoms of local inflammatory process may show themselves during the stage of reaction, such as a diphtheric enterocolitis with foul, purulent, bloody stools; parotid bubo, which goes on to suppuration; or diphtheria of the upper air-passages, with pneumonia and septicemia.

Uremic symptoms may take the place of the two conditions just described, with all the symptoms of an ordinary acute nephritis: large amounts of albumin, free blood-cells, high specific gravity, vomiting, convulsions, and edema; marked dropsy is rare. These symptoms frequently terminate in death. Convalescence, however, may follow the stage of reaction, provided cholera typhoid does not occur.

Occasionally, all these symptoms may be present without the marked serous stools. This condition is known as *cholera sicca*, in which the bowel is paralyzed; hence, its fluid can not be voided. These are extremely fatal cases.

The four following varieties of cholera have been described:

Cholerine, or mild cases.

Cholera fulminosa, a severe form with diarrhea and cramp, patient dying within a few hours with intense toxemia, not passing into the stage of collapse.

Cholera sicca, due to paralysis of the gut.

Cholera toxica, in which the patient is overwhelmed in the course of a few hours from the profound toxemia, and dies before the classic symptoms have manifested themselves.

Diagnosis.—Direct diagnosis is not difficult in the face of an epidemic. In all doubtful cases bacteriologic examination is necessary. In times of epidemics every acute gastro-intestinal catarrh should be regarded as a possible case of cholera, and all necessary precautions taken.

Differential Diagnosis. — Differential diagnosis between cholera nostras and cholera Asiatica may present difficulties, but a bacteriologic diagnosis will soon show the true nature of the affection.

Prognosis.—Prognosis should always be guarded. Cases with mild premonitory symptoms may end fatally. The mortality in epidemics varies between 30% and 80%. Especially unfavorable prognostic influences are alcoholism and the debility of previous disease. The extremes of age bear the disease badly; in individual cases the prognosis is rendered unfavorable in proportion to the intensity of the collapse and the general toxemia.

The death-rate is highest early in an epidemic. The prognosis is greatly modified by treatment. Cases that are promptly treated in the stage of premonitory diarrhea frequently recover.

Treatment.—Prophylaxis.—Complete isolation of the sick, thorough disinfection of all discharges and all articles of clothing, etc., are absolutely necessary. The disease is not contagious, but the specific germ must gain access to the alimentary canal to effect infection. Efficient quarantine must be established. All water and milk should be boiled. No raw fruit or vegetables should be partaken of during the time of the epidemic. Every suspect should be promptly isolated, and all stools disinfected.

Treatment of the Attack.—In the stage of premonitory diarrhea it is considered good practice to clear the bowels at the onset by a prompt laxative. After evacuating the bowel, small doses of calomel may be continued, one-sixth to one-fourth of a grain every second hour. If there be pain and tendency to collapse, morphia should be administered hypodermically. Salol and guaiacol carbonate may be used from time to time as intestinal antiseptics.

In the stage of serous diarrhea the patient should be wrapped in flannels, and external heat applied to the body. Friction of the muscles, if cramp occur, is useful. Fluids should be withheld except a few sips of iced champagne, or small lumps of ice given to allay thirst. The vomiting is often difficult to control ; a mustard plaster over the epigastrium or a few drops of chloroform are useful. If tendency to collapse is great, warm baths (from 103° to 105° F.) may be given. Whisky and brandy are often retained ; if vomited, they should be given hypodermically. Enteroclysis should be practised in the stage of serous diarrhea. It may consist of boiled water at a temperature of 108° to 109° F., to which should be added five or ten drams of tannin, from thirty to fifty drops of laudanum, and some gum arabic ; two liters at a time should be slowly injected into the bowel. The enteroclysis may be repeated at an interval of two hours.

In the stage of collapse the enteroclysis may be repeated, or hypodermoclysis of a normal salt solution may be resorted to. This does not exclude the practice of enteroclysis, which may be used conjointly. Lavage of a very weak acid solution, 1 part hydrochloric acid to from 3000 to 5000 parts of sterilized water, is sometimes useful in allaying vomiting.

Strychnin hypodermically should not be repeated frequently, as the drug may accumulate in the tissues and produce toxic symptoms. Convalescence occupies several days, during which the diet must be carefully regulated.

Cholera typhoid must be treated upon general principles.

THE PLAGUE.

Definition.—An acute, infectious, contagious disease, characterized by inflammation of the lymphatics, with a marked tendency to suppuration, and due to the bacillus pestis.

Synonyms.—Oriental plague; bubonic plague; pest; the black death (of the fourteenth century).

Etiology.—Predisposing Causes.—This is particularly a disease of poverty. Overcrowding, bad hygiene, filth, and insufficient food predispose in regions in which it occurs. It prevails in every climate. Season of the year is without influence. Fear, anxiety, and previous debilitating disease are said to be predisposing causes.

Sex and age are without influence except after the fiftieth year, when it is an extremely rare affection. One attack does not confer immunity.

Exciting Cause.—The exciting cause is the bacillus pestis. (For description of this germ, see p. 119.)

Pathology.—The plague has prevailed in countries in which it has been extremely difficult to make postmortem study; hence, little is known of the pathology.

The spleen is enlarged, swelling, inflammation with a tendency to suppuration of the lymphatic glands, are the constant changes found after death. The inflammation is not always limited to the glands, but may extend to the contiguous tissues, in which extravasation of blood occurs. The inguinal glands are the ones most often involved, although enlargements may be found in the axilla, the neck, and other portions of the body. Petechiæ, ecchymoses, and carbuncles are found upon the skin. The carbuncles often show large sloughs, which are surrounded by an inflammatory process. Parenchymatous degeneration of the heart, liver, and kidneys has been observed.

Period of Incubation.—The period of incubation is from two to five days.

Varieties.—Three varieties of the disease have been described: the *grave*, the *fulminant*, and the *larval* or *abortive* form.

Stages.—The disease has been divided into the following stages for convenience of description:

The stage of prodromes or invasion.
The stage of the fully established disease.
The stage of development of the buboes.
The stage of convalescence.

Symptoms.—Invasion.—The invasion is sudden, with lassitude, pain in the back and extremities; the patient becomes dull and stupid, and acts like a person under the influence of alcohol or a narcotic. Fever may be slight or absent for a time.

Fully Established Disease.—The stage of the fully established disease is ushered in by a chill followed by high fever. The temperature may rise as high as 107° F. or 108° F. The patient becomes delirious, soon passing into stupor and coma. The circulation is rapid and feeble, and symptoms of collapse may occur. The lymphatics now begin to enlarge, and with a sudden fall in temperature, accompanied by copious sweating, the mind clears and the buboes in the groin, armpit, and angle of the jaw become prominent. In a large proportion of the cases the inguinal glands are the only ones affected.

Development of Buboes.—The full development of the buboes marks the third stage. They vary in size from that of a pea to a small orange. As a rule, suppuration takes place, the gland breaking down on the third or fourth day after the formation of the bubo. In grave cases suppuration is absent altogether, carbuncles occurring in a considerable number of the cases. Petechiæ, which often appear early, show themselves in the severer cases.

Convalescence.—In the fourth stage convalescence, which may be greatly protracted by the local lesions, sets in somewhere between the sixth and tenth days. Pus discharging from the lymphatics, carbuncles, and a prolonged relapse may lengthen the disease. Distinct second attacks occur. In the fulminant form death may take place in a few hours.

Prognosis.—The mortality of the plague is greater than that from any other acute infectious disease.

Diagnosis.—In the presence of an epidemic the diagnosis is not difficult. The direct diagnosis would depend upon the prodromes, the high fever with enlargement of the lymphatics, which suppurate, and the protracted convalescence.

Treatment.—Cleanliness and strict observation of hygienic laws are necessary. Quarantine should be of the most energetic kind. The treatment is expectant-symptomatic. A purge at the onset is advantageous. Suppuration should be encouraged in the buboes, and these should be treated by antiseptic surgical methods. Bold stimulation is necessary in the severe cases.

PERTUSSIS.

Definition.—An acute, specific disease, occurring particularly in children, implicating the mucous membranes of the air-passages, characterized by paroxysmal cough. The

disease is highly contagious, and one attack confers immunity.

Synonyms.—Whooping-cough; tussis convulsiva; convulsive cough.

Etiology.—A disease of early childhood, and common between the periods of first and second dentition—that is, between the second and seventh years. It may occur in adults, but is then not well developed and the symptoms are not characteristic.

Period of Incubation.—From seven to ten days.

Exciting Cause.—By analogy it is supposed to be a specific germ, which has not yet been isolated. Epidemics occur late in the spring and early in winter, and last from three to four months, often following or preceding outbreaks of measles or scarlet fever.

Pathology.—There is no specific anatomical lesion. In unfavorable cases pulmonary complications, particularly bronchopneumonia, are present. Pulmonary emphysema, with enlargement of the tracheal and bronchial glands, is found in some cases.

Symptoms.—The disease is divided into two stages—*the catarrhal* and *the convulsive stages*.

Catarrhal.—The catarrhal stage begins as an ordinary acute bronchitis, developing rapidly, with cough that is severe but not at all paroxysmal. Considerable loose phlegm is present at first, and large and small moist rales are heard in the chest throughout the course of the disease. There may be sneezing, slight conjunctivitis, and a temperature between 102° F. and 103° F., with furred tongue, loss of appetite, and constipation. This period lasts from seven to ten days, but is a variable one, and a diagnosis of pertussis can not be made in this stage.

Convulsive Stage.—The symptoms continue, and to the catarrhal is added the convulsive stage. The cough is more violent, paroxysmal, and continuous, until a decided paroxysm takes place. It is a nervous cough, and is often more marked at night, occurring more frequently after taking food, causing the child to vomit.

The paroxysm consists of a volley of coughing efforts that are expiratory in character with one or two short inspiratory acts between them. At the end of the volley of rapid expiratory efforts a long-drawn inspiration occurs with a crowing sound, due to fixation of the glottis but not an abso-

lute closure, and this long drawn inspiration produces the "whoop."

During this period there may be cyanosis, swelling of the veins of the head, producing congestion of the face and neck. Hemorrhages may occur in the upper air-passages, from the throat, nose, tissues about the eyelids, with vomiting and involuntary evacuations. The number of paroxysms during the day vary; there may be from five to ten in the course of twenty-four hours, or there may be as many as forty or fifty. An ulcer under the frenum of the tongue is very likely to occur in this stage, due to the projection of the tongue forward over the teeth in the paroxysm of coughing.

Complications and Sequels.—Hemorrhages appear, such as petechiæ, about the face and neck. Epistaxis and hemoptysis may occur, but they are of little importance. They usually terminate in recovery. Bronchopneumonia is a complication adding greatly to the gravity of the case, being the cause of the fatal issue in many instances.

Pulmonary tuberculosis is comparatively frequent in a considerable proportion of the cases, especially in adolescence.

Diagnosis.—This is impossible in the catarrhal stage, but pertussis can not be confounded with any other disease in the convulsive stage.

Prognosis.—As a rule, it is favorable, the danger being due to complications. The disease usually lasts from four to six weeks.

Treatment.—Isolation is desirable, but is scarcely practicable. Food should be abundant and nutritious, and if the child vomits during a paroxysm, nourishment should immediately be readministered. Antipyrin in doses proportionate to the age of the patient has some influence on the paroxysms. Bromoform may be useful if it can be taken by the child, but it is very likely to give rise to nausea. Good hygiene is essential in the treatment of the case.

PAROTITIS.

Definition.—An acute, infectious, contagious disease, characterized by swelling of one or both parotid glands, with trifling constitutional symptoms.

Synonym.—Mumps.

Etiology.—The exciting cause is an infectious principle, which has not yet been isolated. It is more frequent in the

male than in the female sex. It is commonly encountered in childhood and early adult life, rarely in the extremes of age.

Period of Incubation.—Period of incubation is about two weeks, it may be as long as three weeks.

Symptoms.—Prodromes, if present, are slight, consisting of discomfort, headache, and some fever, with abrupt development of an enlarged parotid gland on one side. Inflammatory edema of the surrounding tissue may occur, so that the ear is pushed upward. The mouth is displaced, and the countenance disfigured. There is difficulty in chewing, swallowing, and eating. The temperature may reach 102° F., but is irregular and not typical. The fever is often absent. In some cases the gland on the opposite side may become affected, but not until several days have elapsed or until inflammation in the other gland subsides. Abscess formation does not occur.

The whole duration of the process is from eight to ten days, prolonged, however, by infection of the testicles when they become implicated, which is more likely to occur in adults than children. Other glands may be affected, such as the sublingual and the submaxillary.

Diagnosis.—Diagnosis depends upon the sudden onset with enlargement in one of the large lymphatics about the neck, slight fever, and rapid subsidence.

Prognosis.—Invariably favorable, all cases recovering.

Treatment.—The patient should be isolated and a mild laxative given at the onset. Rest in bed is important. There is no specific treatment. Attention should be given to the prevention of complications and the alleviation of pain.

INFECTIONS WITHOUT SPECIAL CLASSIFICATION.

TUBERCULOSIS.

Definition.—Tuberculosis is an infectious disease, caused by the tubercle bacillus of Koch, and characterized anatomically by the formation of tubercles.

Etiology.—Predisposing Cause.—This is a wide-spread disease, occurring in all parts of the universe, affecting both man and animals. About *one-seventh* of all deaths are due to this disease. It is more frequent in the tropics than in temperate climates, and more frequent in cities than in the country.

Diseases.	Synonyms.	Exciting Cause.	Period of Incubation.	Pathology.
Influenza.	Epidemic catarrhal fever; la grippe.	Bacillus of Pfeiffer.	From a few hours to two or three days.	Catarrhal inflammation of mucous membranes.
Enteric fever.	Typhoid fever; typhus abdominalis; autumnal fever.	Bacillus of Eberth.	Two to three weeks.	Lesions of Peyer's patch and solitary follicles; enlargement of spleen.
Typhus fever.	Camp fever; jail fever; ship fever.	Not known.	Variable, about two weeks.	Granular and fatty degeneration of muscles and internal organs.
Relapsing fever.	Spirillum fever; famine fever; typhus recurrens.	Spirochæta of Obermeier.	Five to eight days.	Enlargement of the spleen.
Yellow fever.	Yellow jack; black vomit fever.	Bacillus icteroides.	One to seven days.	Fatty degeneration of the liver, kidney, and stomach, with hemorrhages.
Dengue.	Breakbone fever; dandy fever; broken wing fever.	Not definitely determined. McLaughlin's micrococcus. (?)	Two to five days.	Unknown.
Malaria. Tertian fever.	Chills and fever; ague; swamp fever; marsh fever; chagres fever; Roman fever.	Hematozoa of Laveran. Tertian parasite.	About ten days.	Enlargement of the spleen and liver, with pigmentation.
Malaria. Quartan fever.		Quartan parasite.	About thirteen days.	
Malaria. Estivo-autumnal fever.		Estivo-autumnal parasite.	About three days.	
Scarlet fever.	Scarlatina.	Unknown.	Four to seven days.	Often parenchymatous nephritis.
Measles.	Rubeola; morbilli.	Unknown.	About ten days.	
Rubella.	German measles; French measles; rötheln.	Unknown.	About eighteen days.	
Variola.	Smallpox.	Unknown.	From ten to thirteen days.	
Varicella.	Chickenpox.	Unknown.	Ten to fifteen days.	
Erysipelas.	St. Anthony's fire; the rose.	Streptococcus erysipelatis of Fehleisen.	Three to seven days.	
Croupous pneumonia.	Lobar pneumonia; fibrinous pneumonia; pneumonitis; lung fever.	Diplococcus lanceolatus; sometimes other organisms.	Unknown.	Engorgement. Consolidation. Resolution. (Red hepatization; gray hepatization.)
Diphtheria.	Putrid sore throat.	Klebs-Löffler bacillus.	Two to five days.	Pseudomembrane.
Cerebrospinal fever.	Epidemic cerebrospinal meningitis; cerebrospinal typhus; spotted fever.	Diplococcus intracellularis meningitidis.	Three to five days; (?) indefinite.	Inflammation of the membranes of the brain and spinal cord.
Acute rheumatic fever.	Acute inflammatory rheumatism.	Unknown.	Unknown.	
Cholera.	Cholera Asiatica.	Comma bacillus of Koch.	Two to five days.	
Plague.	Oriental plague; bubonic plague; pest.	Bacillus pestis.	Two to five days.	Inflammation of the lymphatic glands.
Pertussis.	Whooping-cough.	Unknown.	Seven to ten days.	
Parotitis.	Mumps.	Unknown.	About two weeks.	

CHART.

Clinical Varieties.	Eruption.	Stages.	Course.	Termination.
Thoracic; gastro-intestinal; cardiac; nervous.			Few days to one week.	Crisis or lysis.
Mild; abortive; ambulatory; infantile remittent; grave; apyrexial.	About seventh day.		From twenty-one to twenty-eight days.	Lysis.
	Fifth day.		About fourteen days.	Crisis.
	Jaundice.	Onset; apyrexia; relapse.	About twenty-one days.	Crisis.
	Jaundice.	Onset; calm; collapse.		Lysis.
	Urticaria; erythema, etc., during stage of remission.		Seven to eight days.	Rapid lysis.
Commonly intermittent		Chill; fever; sweating.	Variable.	Commonly crisis.
Commonly remittent; irregular; continued; and pernicious. (algid, comatose, hemorrhagic.)		Irregular.		
Latens; simple; anginosa; malignant.	End of first or second day.	Catarrhal; eruption; desquamation.	Seven to ten days.	Lysis.
Simple; malignant or hemorrhagic.	Fourth day.	Catarrhal; eruption; desquamation.	Twelve days.	Lysis.
	First day.		Few days.	Lysis.
Discrete; confluent; malignant; varioloid.	Third day.	Invasion; eruption; secondary fever.	Indefinite.	Lysis.
	First day.		About ten days.	Lysis.
	First day.			Crisis commonly.
	Herpes.		Five to thirteen days, more or less.	Commonly crisis.
Pharyngeal; laryngeal; nasal.			Indefinite.	Lysis.
Fulminant; abortive; larval; common.	Herpes and petechiæ.		Irregular.	Lysis.
Ordinary form; cerebral form.			Four to six weeks.	Lysis.
Cholerine; cholera fulminosa; cholera sicca; cholera toxica.		Premonitory diarrhea; serous diarrhea; collapse; reaction.	Short.	Lysis.
Grave; fulminant; larval.		Prodromal; fully established disease; development of the buboes; convalescence.	Indefinite.	Lysis.
		Catarrhal; convulsive.	Four to six weeks.	Lysis.
			Short.	Lysis.

Age, Race, and Sex.—All races and ages are susceptible, but the negro and Indian are particularly prone to this affection. The Hebrew race possesses some degree of immunity. Of the two sexes, the female is probably more frequently affected.

Occupation. — Certain occupations predispose, especially those that confine individuals to indoor life, depriving them of sunlight and necessary exercise, or exposing them to dampness.

Infected dwellings predispose to the disease. It frequently reappears in certain localities in cities, and in houses in which tubercular subjects have died.

Heredity. — Hereditary predisposition to the disease is marked, but the direct transmission of the specific cause does not occur.

Individual Predisposition.—Individual predisposition consists largely in the shape of the chest. The long, narrow, flat chest, with depressed sternum is the form most frequently met with in tubercular patients. This may indicate nothing more than a delicacy of constitution with incomplete growth and imperfect development, but this form of chest, known as the "expiratory" form, is almost invariably met with in chronic tuberculosis. There must be some predisposition so that the germ may find a suitable soil for growth.

Exciting Cause.—A bacillus discovered by Koch in 1882. This organism is found universally distributed in the dust, in soil, and in the various diseased tissues. It is found both in man and animals. (For description, see p. 104.)

Modes of Infection.—Inoculation.—The inoculation of tubercular material directly through the skin produces tuberculosis. This process is extremely rare in man, and is only met with in those who come in contact with the dead or with the products of dead animals.

In such instances local tubercular processes are formed that may exist for years without showing the slightest tendency to spread. They should be classed with cases of lupus, of which they are practically a variety.

Infection by Inhalation.—While it has been shown that the expired air of consumptives is not infective, the virus being contained in the sputum, the danger lies in the sputum becoming dried, mixed with dust, and inhaled by a susceptible person. In this way the disease is most frequently transmitted.

Infection through the Gastro-intestinal Tract.—Infection through the gastro-intestinal tract may occur as a result of drinking infected milk, this being derived from a tubercular udder, or by infected meat. Again, infection results from swallowing tubercular sputum, the latter being the most frequent cause of intestinal tuberculosis.

Pathology.—*The naked-eye appearance* of a tubercle is as follows: They are small nodules, varying in size from that of a millet to that of a mustard seed, and sometimes larger. They are multiple, distributed uniformly, or clustered, and show a tendency to fuse together, forming large masses. They are generally quite firm, gray in color, and when caseation takes place, the centers are more or less of a creamy yellow color. The tubercles are easily removed by dissection.

The pathology of the tubercle is the same in all organs.

Microscopic appearance: After the bacillus lodges in an area, there is proliferation of the connective-tissue cells, which group around the irritant. Some polynuclear leukocytes wander into this area, but the latter feature is not marked. The accumulation of cells causes a nodule, which may be termed a "young tubercle." One or more of the connective-tissue cells near the center of the mass may become enlarged, the nucleus proliferating and grouping around the periphery, called a "giant cell." The cells next to the giant cells seem to enlarge somewhat, and are called "epithelioid" cells, and the outermost layer of cells is called "lymphoid" or round-cell area. The central part of the mass in almost every case undergoes cheesy necrosis, probably as a result of the cutting off of the circulation, and also the influence of the toxin. The cheesy mass, giant cells, epithelioid and small cells, arranged in the way just described, constitute a full-grown tubercle. These tubercles show a tendency to spread in all directions, more particularly in the route of the least resistance. Nature frequently shows a tendency to limit the spread by the formation of fibrous connective tissue around the tubercle, called a "healed tubercle." (Some of the connective-tissue cells of the outer layer form fibroblasts, and finally fully formed fibrous connective tissue develops.) Infiltration of lime salts into this capsule is common.

It is stated by some pathologists that occasionally the giant cells that are nearest the center of the tubercle throw out fibrillated ends, and this process continues still further until fully formed fibrous tissue results. This explains the

entire cure in some instances. This view, however, is not accepted by most authorities.

Secondary infection from a primary focus sometimes occurs. The tubercle breaks into a blood-vessel, the material being carried throughout the circulation, and lodges in various tissues, causing wide-spread or disseminated tuberculosis. In this condition the tubercles are rarely larger than a millet seed; hence the name, *general miliary tuberculosis.* They are usually uniformly of this size, and rarely show marked caseation. By many it is considered that a tuberculous lesion never causes suppuration, and that if the latter occurs it is as a result of secondary infection by some of the pyogenic micro-organisms. This condition is met with particularly in pulmonary lesions.

Various tissues and organs are especially prone to the infection, as the lung and the lymphatic system. The spleen, kidneys, liver, intestines, and the brain are also very liable, but to a less extent than the tissues already indicated. The muscular and fibrous tissues are more or less exempt, but in rare instances may be affected.

Amyloid disease sometimes results as a consequence of tuberculosis. Almost invariably fatty infiltration of the liver is associated with the chronic pulmonary forms.

ACUTE MILIARY TUBERCULOSIS.

Etiology.—As has already been indicated, when tubercle bacilli are distributed throughout the body by the blood stream, there is general infection, so that the picture may be one of acute infection. This is commonly the result of the breaking down of an old lesion, which may be in any part of the body, frequently in a lymphatic gland, in a pulmonary lesion, or in the bone-marrow. Rarely at the autopsy can this primary focus be found. It has been claimed that general miliary tuberculosis sometimes develops as a primary infection.

The distribution of the tubercles in this variety is unequal, being abundant in some organs and scanty in others.

Clinical Varieties.—1. The General or Typhoid Form.—In this variety the resemblance to an infectious disease is striking, and the course and general features may closely simulate enteric fever. Indeed, the resemblance may be so complete that days, and even weeks, may elapse before a positive diagnosis can be made—perhaps not until the autopsy.

Symptoms.—The patient generally shows signs of failing health, with anorexia, accompanied by headache, chilly sensations, slight cough, and digestive disturbances. These symptoms may last for a week or two. Even epistaxis has been noted in such cases. Gradually the patient becomes feverish, with dry tongue, rapid pulse, respirations increased, and pulmonary symptoms becoming more marked. The pulse, however, is rarely, if ever, dicrotic. The temperature does not show the steady increase so typical in the first period of enteric fever. The remissions are greater, and the entire fever-curve is more irregular. The fever may be intermittent, and in the early morning hours the temperature may be normal or subnormal. At times the inverse type of temperature is present. In rare cases pyrexia does not occur at all. Delirium is an early symptom, and is accompanied by restlessness, subsultus tendinum, and carphology. There is cutaneous hyperesthesia. Albuminuria is almost constant, and the urine often gives Ehrlich's diazo reaction. The gastric symptoms consist of vomiting, diarrhea, and even enterorrhagia, if tubercular ulcers exist in the bowel. The abdomen is tympanitic, and the spleen is enlarged. The disease may last from a few days to five or six weeks. The patient generally dies in coma, with Cheyne-Stokes respiration. The disease is invariably fatal. Tubercle bacilli have occasionally been found in the blood and in the dejecta.

2. The Pulmonary Form.—In this variety the symptoms are very nearly the same as those already enumerated, with the exception that they are more closely related to the pulmonary system. There may be spitting of blood, although this is by no means the rule. The cough is more marked, the expectoration more profuse, and the respirations more rapid. Tubercle bacilli are rarely found in the sputum. When present, they are of diagnostic import.

Physical Signs.—The physical signs may be those of a rapid breaking up of the pulmonary structure, with all the physical signs of bronchopneumonia.

3. The Meningeal Form.—This variety is found most frequently in children, and by the older writers was called "dropsy of the brain."

The distribution of tubercles in this variety is particularly in the membranes at the base of the brain and in the Sylvian fissure; hence the symptoms closely simulate meningitis. The onset is variable; it may begin abruptly, with intense cerebral

excitement, or there may be symptoms of mania, and it may in a few days prove fatal.

In a large proportion of cases, however, the disease runs a subacute course, and may be prolonged over a period of months.

The course of the disease has been divided into several stages, which are sometimes well defined: *a prodromal stage, a period of excitement*, and *a period of paralysis.*

Prodromal Stage.—The disease may occur after one of the eruptive fevers, or may follow an injury such as a fall. The child becomes irritable and restless, especially at night; there are loss of appetite and change in its disposition. Headache, tired feelings, pain in the limbs, accompanied often by obstinate vomiting, nausea, and constipation take place. The disease rarely begins with a convulsion. Fever is absent in the prodromal stage.

Stage of Excitement.—The second stage is marked by an aggravation of the symptoms just enumerated, with the addition of fever. The pain in the head becomes intense, the face is flushed, and the child utters a short, sudden cry known as the *hydrocephalic cry.* The headache may be frontal, though it is usually general, being aggravated by rays of light, noise, or any slight movement. The screaming may be continuous, only ceasing when the child is exhausted. Vomiting is common, and may be independent of the taking of food; it generally lasts but a short time, desisting in a few days; occasionally, however, it remains throughout the entire course of the disease. Obstinate constipation is characteristic. The fever, from which the onset of this stage may be reckoned, is at first moderate, with evening exacerbations, being about 102° F., with rapid pulse, ranging from 120 to 160 a minute. The abdomen is prominent, the tongue furred, and the breath offensive.

Nervous symptoms are present, most often delirium. The pupils are contracted, and strabismus may be noted. There is marked cutaneous hyperesthesia, and the reflexes are exaggerated. The "tache cérébrale" occurs, but is not diagnostic. Convulsive movements are common. There may be tonic spasm. If the spinal meninges are involved, there may be opisthotonos. This period persists for a week or ten days.

Stage of Paralysis.—In the final period, "the stage of paralysis," the fever becomes higher, often reaching 105° F. or 106° F.; there may be hyperpyrexia, the temperature

rising to 110° F.; spasmodic contractions are frequent, and there are tremor and twitching of the tendons and muscles, with local paralysis. In this form the ocular features are prominent. There may be conjugate deviation of the head and eyes. *The third nerve is most frequently involved*, causing ptosis. Strabismus and optic neuritis are met with. Tuberculosis of the choroid has been observed. The duration of the disease is variable, the majority of cases lasting about three weeks, but when this form occurs in adults it may be prolonged to sixteen weeks or longer. The disease is almost invariably fatal. Leukocytosis has been found throughout the course of the disease.

Differential Diagnosis.—Differential diagnosis must be made between miliary tuberculosis and enteric fever.

Enteric Fever.	*Miliary Tuberculosis.*
Typical curve of the temperature.	Temperature-curve atypical; may be inverted type of temperature.
Pulse not particularly rapid.	Pulse always accelerated, especially in the meningeal form.
Enlarged spleen early.	Enlargement of the spleen occurs, but spleen can rarely be palpated.
Eruption at end of first week, running a typical course.	Eruption rare and never typical.
Bronchitis occurs over the entire lung, more particularly marked at lower than upper parts.	Signs of bronchitis with particular intensity at the apices of the lung showing early bronchopneumonia.
Pericardial and pleural friction sounds rarely present.	Pericardial and pleural friction sounds frequently present.
Presence of the typhoid bacillus in excreta; sputum free from tubercle bacilli.	Tubercle bacilli in the sputum and in excreta, although may rarely be found.
Diarrhea common; stools may contain typhoid bacillus.	Constipation usually the rule. Tubercle bacilli may be present in stools.
Tongue characteristic. Dicrotic pulse occurs.	Dicroticism rare; tongue atypical.
Meningitis extremely rare.	Meningitis common.
Widal reaction present.	Widal reaction absent.

TUBERCULOSIS OF THE LUNGS.

Synonyms.—Phthisis; consumption.

Varieties.—Three varieties are described: *Acute pneumonic phthisis*, *chronic ulcerative tuberculosis*, *fibroid phthisis.*

ACUTE PNEUMONIC PHTHISIS.

The disease is apt to attack persons who have been weakened by previous illness, exposure, or dissipation. It may, however, occur in persons in perfectly good health. In nearly all in-

stances the disease is secondary to a preexisting tubercular focus, most often of the lung.

Pathology. — Small tubercles are thickly distributed throughout both lungs, occasionally only confined to one, and rarely to a single lobe, and show slight tendency to the formation of cavities. The lungs in this condition may weigh as much as 1600 grams. Occasionally, we find caseous bronchopneumonia.

Clinical Varieties.—Clinically, two varieties have been differentiated: (1) the pneumonic form and (2) the bronchopneumonic form.

Pneumonic Form.—The disease often begins abruptly, with chill, following exposure. The temperature rises suddenly, with all the initial symptoms of an ordinary croupous pneumonia, and with cough and pain in the side. The expectoration may be blood-tinged and typically pneumonic, rusty sputum showing itself. In other cases the disease commences more gradually. One lobe may be found first affected, and the stage of consolidation come on with extreme rapidity; the process spreads rapidly from lobe to lobe, so that soon the entire lung is involved. The fever is high and persistent, the pulse rapid—from 120 to 140 a minute. As the extension of consolidation proceeds, the respirations become accelerated, but even with all these physical conditions dyspnea and cyanosis may be absent.

About the ninth day of the disease, instead of crisis taking place, the fever persists and becomes irregular; the expectoration is less rusty and becomes mucopurulent or of a greenish color. Occasionally, expectoration may be absent altogether.

Hemoptysis may occur at any time during the course of the disease. The spleen is enlarged, and there may be edema of the lower extremities. Nervous symptoms are present, but are usually proportionate to the fever. The course of the disease is variable, death generally ensuing in about six weeks. In some instances death takes place early, or the disease may not prove fatal for five or six months.

Bronchopneumonic Form.—In this form persons in good health are rarely affected, and the disease presents a typical picture of *galloping consumption.*

Hemoptysis sets in early; loss of weight is rapid; there are pronounced chills, sweats, and intermittent fever. The pulse is rapid, and the cough distressing. The apices of the lung

are most frequently involved. The signs at first may be very slight; subsequently, as the involved areas coalesce, bronchial breathing and dullness upon percussion are prominent. The disease may prove fatal in from six to twelve weeks; the majority of cases of "galloping consumption" conform to this type. Occasionally, in the most desperate cases, when all hope has been abandoned, the symptoms ameliorate and the disease becomes chronic. Tubercle bacilli may be present in the sputum.

CHRONIC ULCERATIVE TUBERCULOSIS.

Synonyms.—Phthisis; pulmonary consumption; chronic phthisis. This form embraces by far the largest number of cases of pulmonary tuberculosis.

Pathology.—The lesions in this variety are variable. The most frequent seats of involvement are the apices of the lungs. The disease first begins as a catarrhal inflammation of the finer bronchial tubes and alveoli, the bacillus next setting up irritation in the interstitial substance of the lung. Tubercles are formed, and caseous degeneration appears. The nodules multiply, the primary seat becoming densely infiltrated. The tubercles coalesce, and large cheesy deposits are formed; as the large bronchial tubes become involved and the mass is discharged, cavity formation takes place. The blood-vessels show great resistance to the action of the poison, therefore vessels stretching across cavities as tense fibrous bands are sometimes found. In a paroxysm of coughing the vessels may be torn and copious hemorrhages result. Occasionally, slow involvement of the vessel-wall occurs. This is especially true of the smaller blood-channels. The pleuræ are almost invariably involved, as are also the peribronchial glands. The pericardium may show tubercular lesions. There is frequently encapsulation of the tubercles, especially if there are lesions at the apices.

Mixed infection is present in long-standing cases, causing suppuration and ulceration. The latter process may extend into the pleural sacs. If free communication between the bronchi and these cavities is established, pneumothorax may result. Fatty infiltration of the liver is almost constantly present, amyloid disease rarely.

Symptoms.—No disease presents so varied a symptomatology at the onset as chronic pulmonary tuberculosis. The disease may begin in one of five ways:

1. It may begin insidiously, with loss of strength and flesh,

impairment of appetite, and slight evening rise in temperature. There may be some anemia and functional derangement of the digestive system.

2. After repeated attacks of bronchitis, and in many cases after an attack of influenza.

3. The symptoms may resemble an ordinary pleurisy. This may be of the dry form, or pleurisy with effusion, the other symptoms following the pleuritic attack.

4. The onset is marked by profuse hemorrhage, the other symptoms following this condition.

5. It begins as a laryngitis, with hoarseness and loss of voice, remaining so for some time before there is involvement of the lung.

Cough.—The most important symptom is cough, which is dry and short, but accompanied sooner or later by expectoration. There is no relation between the severity of the cough and the gravity of the disease. When the larynx is involved, the cough is particularly hoarse and muffled, and may be paroxysmal in character: it may be so incessant as to produce vomiting.

Expectoration.—At first the expectoration is scanty, but soon becomes mucopurulent and viscid; it may be thin and watery from an admixture with saliva; later in the disease it collects in thick lumps of a yellowish color—"nummular sputum." This is common where cavities are formed in the lung. In the advanced cases it becomes opaque, thick, and of a yellowish color. Blood is often found admixed with it. Fetor of the sputum is extremely uncommon, such changes being due to a mixed infection. It can not be said that any variety of sputum is pathognomonic of this disease. Only by the microscope can the diagnosis be made certain, when the presence of tubercle bacilli have been demonstrated in the sputum.

Hemorrhages.—Hemoptysis is a prominent symptom of the disease, and may vary from a few drops to a pint or more; this may prove directly fatal if the hemorrhage is very large. It is, however, more common for the patient to recover from the direct consequence of blood-spitting. Hemoptysis may be repeated frequently, and last from hours to days, with intermissions.

Dyspnea.—This is rarely complained of, except in the later stages of the disease. Increased frequency of respiration occurs early, upon slight exertion, being greater toward evening.

Pain.—Pain in the chest is due to pleural involvement; it may, however, be of muscular origin, due to the violence of the cough.

Fever.—Fever, next to the cough, is the most important symptom; it must be ascribed to the presence in the blood of a toxin produced by the bacillus or accompanying micro-organism. The fever is usually high toward afternoon and evening. A slight evening rise may be one of the first symptoms. The onset of the disease may be marked by slight shivering, but a decided chill is present only, as a rule, in acute cases. The inverse type of temperature is sometimes present in this disease. Two main forms of fever may be distinguished: the intermittent and the remittent; either may predominate, but usually there is a combination of both. The irregular course of the fever is diagnostic. Subnormal temperature not infrequently occurs in the morning; profuse night-sweats are a common symptom, and may have no relation to the intensity of the fever.

Emaciation.—Emaciation is profound and proceeds from day to day; hence the names "phthisis" and "consumption."

Anemia.—Anemia is marked, often of the chlorotic type, with no increase in the leukocytes, excepting when there is mixed infection.

Pulse.—The pulse is rapid, but often small, being higher in the evening.

Skin.—The skin of the tubercular patient is usually oily, and pityriasis is common; the pigmentation may be so marked as to simulate Addison's disease. Cyanosis is rare; coldness of the extremities is a common complaint.

Lupus is occasionally found. The clubbing of the fingers and toes occurs in tuberculosis, being also found in other forms of disease in which failure of nutrition is prominent. The hair becomes thin and straight, but may remain quite thick and unusually profuse. On some persons a fine down is noticed all over the body. Edema of the feet is an almost constant symptom in the later stages of the disease.

Gastro-intestinal Symptoms.—Anorexia, vomiting, and nausea are symptoms from the onset, but are especially marked in the later stages. Diarrhea is a very serious symptom. It may occur early, but usually takes place later in the disease, and is associated with ulceration of the bowel. Tubercular disease of the stomach is rare.

Nervous Symptoms.—It has been known from the earliest writings that the peculiarity of the tubercular temperament consists in the marked hope of recovery. Patients with far-advanced disease frequently make plans for the future and confidently expect recovery. Other nervous symptoms are rare in chronic tuberculosis.

Physical Diagnosis.—Inspection.—Great attention should be given to the shape of the chest, which in tubercular subjects is long and narrow. The intercostal spaces are wide, the ribs are more vertical in direction than normal, and the costal angle is very nearly acute. The chest is flattened in its anteroposterior diameter, the sternum is depressed, and the costal cartilages on one or both sides are prominent. The lower sternum may present a deep cavity, the so-called "funnel breast." The two sides of the chest may be unsymmetric. In the early stages the clavicular regions show no marked changes, but if the disease in the apex has persisted for some time, changes are noted.

The clavicle on the affected side is prominent, the supraclavicular and infraclavicular spaces are distinct, and there is marked flattening, corresponding to the first, second, and third ribs of the affected side. In advanced cases the intercostal spaces are much narrower, and the diseased side is considerably shrunken. The region corresponding to the heart may show a wide area of impulse.

Defective expansion, especially of the apex of the lung, is an early and distinctive sign of tuberculosis. While most cases of tuberculosis show these changes, it must be borne in mind that advanced signs of tuberculosis may be present without any change in the contour of the chest.

Palpation.—Palpation confirms inspection, and if disease of the apices be present, increased vocal fremitus will be noticed here as well as in other affected areas. At the bases a pleural exudate, which may complicate the disease, gives diminished or absent vocal fremitus.

Percussion.—With deficient expansion at the apex there is usually some impairment of resonance; this may show early. The note is higher in pitch than normal, and may be quite dull upon percussion if the disease is advanced. If cavities occur, the dullness gives place to hyperresonance, or even tympany.

Auscultation.—Prolonged expiration is an early and valuable sign. On the other hand, the first noticeable change

may be a harsh, rude, inspiratory murmur, the inspiration being jerky and of the so-called "cog-wheel" rhythm. If consolidation be marked, bronchovesicular and even tubular breathing may be present. Rales are heard, and may be due to an associated bronchitis, occurring only upon deep inspiration or during paroxysms of coughing. As the disease advances, however, crepitant and subcrepitant rales appear. Pleuritic friction may exist at any stage, but is especially likely to occur when cavity formation takes place. The so-called "cardiorespiratory" murmur is often present; it is best heard during inspiration and in the anterolateral regions of the chest.

Signs of Cavity.—If the cavity exists at the apex, retraction in the infraclavicular region becomes prominent; this is combined with immobility of the affected side. The vocal fremitus is much increased, and the percussion sounds may be those of hyperresonance or tympany. The tympanitic quality and the increased vocal fremitus disappear when the cavity is filled with fluid. *Wintrich's sign* may be present—a distinct change of pitch when the mouth is opened or closed. Alteration in deep inspiration or deep expiration with a change in the position of the patient may occur. If the cavity be large, with thin walls, superficially situated, and connected with the bronchus, the "cracked-pot" sound is present upon percussion. On auscultation over a cavity great change in the breath-sounds takes place: they may be cavernous, tubular, or amphoric. In large cavities both inspiration and expiration may be typically amphoric. Over the area of a large cavity no breath-sounds may be heard. This may be due either to complete filling of the cavity with fluid or to the blocking of one of the bronchial tubes by a tough plug of mucus. The rales heard over the cavities are coarse, and may be bubbling in quality; they are generally increased by deep inspiration or by coughing.

The vocal fremitus is increased over a cavity if it be empty and superficially situated. Whispering pectoriloquy is present under these circumstances. If the cavity be near the heart, the sound of that organ may be heard, being directly transmitted.

Complications.—The larynx may be the seat of extensive disease, with symptoms of huskiness of the voice, pain on swallowing, and wheezing cough. There may be aphonia. Ulcerative conditions may also be present.

Emphysema may be present in parts of the lung not affected by the tubercular process. Gangrene of the lung is rare. Pleurisy early accompanies chronic tubercular processes. It is most common in the apices, the signs of friction being prominent. Pleurisy with effusion usually precedes chronic pulmonary tuberculosis; adhesions from pleurisy may occur at any part of the lung and become permanent. Pneumothorax is a common complication late in the disease, and rapidly proves fatal.

Endocarditis is by no means so rare as was formerly supposed. Tuberculosis of the kidneys and bladder sometimes results during the course of the disease, giving the characteristic symptoms due to these conditions.

Diagnosis.—In advanced cases the diagnosis may be made with ease. If puzzling symptoms appear, the detection of the bacillus will determine the diagnosis.

FIBROID PHTHISIS.

Pathology.—The lung showing fibroid phthisis reveals a small organ, greatly contracted and indurated, and on microscopic examination tubercles will be found, surrounded by fibrous connective tissue. In this condition the entire lung tissue may be replaced by tubercles with a great amount of fibrous connective tissue. It has been noted that one lung will show this condition while its fellow remains apparently normal, or compensatory hypertrophy will result.

Symptoms and Physical Signs.—The symptoms of ordinary ulcerative tuberculosis may for a long time precede the development of the fibroid condition. The marked changes begin with the temperature, which, having been higher in the evening, now becomes normal; sweating ceases, and the patient regains some degree of strength. Upon inspection it will be found that the affected side becomes more and more flattened, the intercostal spaces retract, and the respiratory movement diminishes. Impairment of resonance is noted on percussion, until complete dullness may be distinguished. With these signs there is displacement of organs, due to adhesions upon the affected side: the heart is pulled toward the diseased side, and the stomach may be drawn up. Emphysema of the opposite lung and hypertrophy of the heart develop. Bronchial breathing is marked, and pectoriloquy is often present. The respirations show little alteration, but the cough is paroxysmal and may be attended with profuse expec-

toration. The urine often becomes albuminous. Hemoptysis is very much more marked in this form than in any other variety.

The disease may remain stationary for years, but hemorrhages are extremely likely to occur. The principal dangers in fibroid phthisis are: the hemorrhage, which is often profuse; the gradual extension of the disease; and the failure of the circulation from cardiac influences. Dropsy, especially of the feet, takes place in the later stages of the disease.

Diagnosis.—This is difficult. The history is of importance, as the disease frequently follows pleurisy, bronchopneumonia, or chronic bronchitis. The absence of fever is an important diagnostic sign. The face is characteristic, presenting the most marked features present in tubercular disease. The complexion is muddy, the hair is thick, and the eyelashes are long. The presence of tubercle bacilli is important in the diagnosis.

Intercurrent Diseases.—The most important of these are lobar pneumonia, erysipelas, endocarditis, and anal fistula. The latter is quite common in pulmonary tuberculosis.

TUBERCULOSIS OF THE LYMPH-GLANDS.

Tubercular adenitis occurs at all ages, but is most frequent in children. It is common among the poor. The negro is especially susceptible. It is frequently associated with coryza, eczema of the scalp, and conjunctivitis.

Pathology.—Tuberculosis of the lymphatic glands differs little in its pathology from the condition in other parts of the body, the most frequent seat being the submaxillary lymphatic glands, usually unilateral; if bilateral, the enlargement is greater on one side than upon the other. Other glands, such as the anterior and posterior cervical chains, the axillary, inguinal, mediastinal, and mesenteric, may be the seats of this affection.

Suppuration results early, and the glands mat together. It is an undecided question as to whether the pus is the result of the tubercle bacillus, but it is generally supposed to be due to a mixed infection.

Healing by encapsulation frequently results. In this event it is spoken of as being a drawn battle between nature and the disease. The focus for future infection may always be opened, and may give rise to general miliary tuberculosis or recurrence of the old condition.

Clinical Forms.—The clinical forms may be classified under two headings: *generalized tubercular lymphadenitis* and *local tubercular adenitis.*

Generalized Tubercular Lymphadenitis.—In generalized tubercular lymphadenitis the lymphatic system may be exclusively affected, and this is, perhaps, a more common type than is usually supposed. It is found in both children and adults. The symptoms are those of general cachexia, with little fever and without distinctive signs of involvement of the lung or abdominal organs. There may be irregular fever, and the patient may die without involvement of any of the viscera. These cases closely simulate Hodgkin's disease.

Local Tubercular Adenitis.—This may affect the cervical, tracheobronchial, mesenteric, peritoneal, or other groups. It is usually considered to be a surgical affection, most common in young individuals, and not necessarily in those of a tubercular diathesis.

The disease runs an extremely chronic course, and the glands are very likely to suppurate, surgical interference then being imperative.

(For differential diagnosis between this disease and Hodgkin's disease, which it sometimes closely resembles, especially in its onset, refer to Hodgkin's Disease, p. 625.)

TUBERCULOSIS OF THE SEROUS MEMBRANES.

Under this heading will be considered the pleura, the peritoneum, and the pericardium.

The Pleura.—The pleura, as has previously been mentioned, may show the first sign of tubercular infection. If effusion follow, it may be serous, serohemorrhagic, or purulent; of these conditions the serous effusion is the most frequent. It is conceded by bacteriologists that tubercular effusions are usually sterile, except when there is a mixed affection.

The onset of the pleurisy may be insidious. Occasionally, the disease may come on acutely and run a subsequent chronic course.

Symptoms.—The symptoms and signs in a tubercular effusion are those found with pleurisy resulting from other conditions, and are considered under that head. (See p. 413.)

Tuberculosis of the pleura may occur as a primary condition, frequently as a secondary affection, from tuberculosis of

the lung involving the visceral layer of the pleura or in the course of general miliary tuberculosis.

Tuberculosis of the Pericardium.—This may be of either the acute miliary or the chronic form. It is met with at all periods of life, and is most frequently found in the miliary variety. Effusions may be met with in the acute form, but are more commonly found in the chronic; they may be serous, hemorrhagic, or purulent; in the majority of cases they are serous.

Symptoms.—The signs and symptoms are the same as those in pericardial effusion occurring under other conditions. The condition is rare. (See p. 316.)

Tuberculosis of the Peritoneum.—The disease appears at all ages, but is more common in childhood. Males are more frequently attacked than females, and it is more often met with in the negro race than in the white race. It is also found associated with intestinal or mesenteric tuberculosis.

The condition may be primary in the peritoneum, and may be confined solely to this membrane. A common mode of infection is through the intestines; this may also occur through the lymphatics or by extension from the pleura or the pericardium. It is often of the miliary variety, but also of the chronic ulcerative and chronic fibroid forms.

Exudation is common, and may be serous, hemorrhagic, or purulent. The serous exudate is the most often encountered.

Symptoms.—The symptoms are those of peritoneal effusions in general. The onset is gradual, with or without fever. The patient soon becomes anemic. Nodules may be felt throughout the abdomen, these being the tubercles upon the peritoneum. It is commonly regarded as a surgical affection. (For symptoms of peritoneal effusion see p. 510.)

TUBERCULOSIS OF THE ALIMENTARY CANAL.

This may affect the lips, but that is a rare situation. An ulcer may occur, and is more common in association with laryngeal and pulmonary disease.

The diagnosis must often be made by inoculation experiments, or by finding the bacillus of tuberculosis in the ulcer or in tissues removed for microscopic examination.

TUBERCULOSIS OF THE TONGUE.

This is nearly always associated with tuberculosis of other organs, and is most commonly at the dorsum, although the tip may be involved. It may occur from direct extension from the pharynx or epiglottis.

Palate.—The palate is only involved from extension. The salivary glands apparently show a decided immunity against tubercular infection; extremely few cases have been reported.

The tonsil may be the seat of tuberculosis. Usually, however, tubercular disease is found elsewhere in association, most commonly in the lungs.

The pharynx and esophagus are likewise rarely involved, but usually from extension.

TUBERCULOSIS OF THE STOMACH.

Tuberculosis rarely affects the stomach, being either of the miliary or chronic caseous variety. The disease is secondary. The pyloric extremity and the greater curvature are usually involved. Perforation from a tuberculous ulcer has been recorded.

TUBERCULOSIS OF THE INTESTINES.

This, with the exception of enteric fever, is the most frequent cause of intestinal ulceration. The large and small intestines may be affected, either in the course of the general miliary process, affecting usually the peritoneum in this condition, or as a result of infection through the gastro-intestinal tract, resulting in the chronic ulcerative variety. The tubercular ulcer is the result of chronic caseous tuberculosis. Any part of the large or of the small intestine may be the seat of this condition, but usually the ileum is most prominently affected. The infection starting in Peyer's patch, anatomic tubercles are formed, and their spread is marked by a tendency to lateral distribution (in the short axis of the bowel).

As caseation extends, rupture occurs into the lumen of the bowel and the ulcer is formed. Its characteristics are the following: It is irregularly oval, the greater diameter of the ulcer being in the short axis of the bowel: the edges are overhanging, and the peritoneal coat is thickened as a result of fibrous connective-tissue formation. Perforation is rare. The

intestines, as a rule, do not show hyperemia around the affected area, but upon closer examination when the intestine is held toward the light, newly formed tubercles may be seen scattered throughout the intestines.

Hemorrhage may occur from erosion of a vessel in the course of the development of the ulcer.

TUBERCULOSIS OF THE LIVER AND PANCREAS.

Tuberculosis also occurs in the liver and pancreas. In the liver it is usually of the miliary variety, the distribution of the tubercles being quite general. The tubercles are small, and *often bile-stained.* Chronic caseous tuberculosis of the liver is rare, and usually secondary to tuberculosis of the peritoneum.

Tuberculosis of the pancreas is rare.

TUBERCULOSIS OF THE GENITO-URINARY SYSTEM.

Tuberculosis of the genito-urinary system is rare, and the process usually involves several parts of the tract. It may be either miliary, or caseous—primary or secondary. It is commonly considered a surgical affection.

TUBERCULOSIS OF THE KIDNEY.

The miliary form does not produce symptoms that are diagnostic. In the caseous form the symptoms are extremely variable, but are usually those of pyelitis. The urine may be purulent, or contain flaky masses of caseous material for a considerable time, although the patient may have little or no distress.

Polyuria may be present, with aching pains in the loins; hemorrhages are not uncommon. The disease is frequently unilateral; however, as it advances, both organs may be affected.

Irregular fever, with loss in weight and strength accompanied by rigors, is common. The urine presents changes common to pyelitis, pus-cells, blood, epithelium, and caseous masses being found.

The tubercle bacillus is found in the urine. The smegma bacillus is frequently a source of error. (For mode of differentiation see p. 105.)

TUBERCULOSIS OF THE MAMMARY GLAND.

Tuberculosis of the mammary gland is met with and is a surgical affection.

TUBERCULOSIS OF THE BRAIN AND CORD.

Tuberculosis of the brain of the miliary form has been described under acute miliary tuberculosis. The caseous variety is sometimes found, but is very rare. The symptoms are not diagnostic, other than the signs of a general localization of a tumor. When symptoms of this sort occur in children, with tubercular lesion elsewhere, this condition should be suspected.

Tuberculosis of the spinal cord appears in the same forms as in the brain.

TUBERCULOSIS OF THE ARTERIES AND HEART.

Tuberculosis of the arteries and heart (endocardium and myocardium) rarely occurs.

Diagnosis of Tuberculosis.—The diagnosis of tuberculosis depends upon the finding of the bacillus, or inoculation of tubercular material in animals, producing the disease, or injection of tuberculin, producing reaction when the disease is present, as is now so frequently practised in veterinary medicine. The diagnosis is difficult when the bacillus is not found, especially so in the miliary variety.

Prognosis.—Spontaneous recovery from local tuberculosis is not infrequently met with, and even tubercular disease of the bones, of the joints, and adenitis may heal without medication. Postmortem examination shows that a very large percentage of the bodies examined present tuberculous foci either in the lungs, the glands, or elsewhere, which have healed in. Cure sometimes takes place even in advanced cases of tuberculosis, as has been shown in cases in which all the symptoms of a marked chronic tubercular process have been present, with bacilli in the sputum, which have afterward disappeared and the patient regained complete health.

The following points are of favorable prognostic import in average cases. Previous good health, good family history, good digestion, and favorable environment. A slow onset without much fever and absence of great consolidation is also favorable in tuberculosis of the lung.

The cases that begin with pleurisy run a very protracted course. Among the favorable symptoms, the absence of fever is the most important. The higher and more persistent the fever, the graver the prognosis. Repeated attacks of hemoptysis are unfavorable, particularly if high fever is also present.

Probably the gravest cases of the chronic pulmonary forms are those that begin with early gastric disturbance. In chronic cases the average duration of tuberculosis is about two years.

Treatment.—Prophylaxis.—The most important measures of prophylaxis are absolute and total disinfection of the infected excreta, particularly the sputum. As has already been pointed out, when the sputum dries and is carried by dust particles, and again inhaled by a susceptible person, infection arises. The majority of infections occur in this way. Veterinary inspection of milk and meat is of great importance.

Individual Prophylaxis.—A tuberculous mother should not be allowed to suckle her infant. The convalescence of children from fevers, particularly measles and whooping-cough, must be carefully guarded. It is not well for a healthy person to sleep with one suffering from tuberculosis.

Persons subject to tuberculosis should always endeavor to obtain outdoor occupation.

Dietetic Treatment.—Blood-producing foods, which are easily digested, such as red meats, milk, eggs, fats, etc., are of especial use in the treatment of tuberculosis. It is important that the digestion be preserved and especial attention paid to it. The meals should not be large, but frequently repeated, so that the patient may receive food every few hours. Alcohol is not necessary as a routine treatment.

Climatic Treatment.—Climatic treatment is of great importance, an even, dry climate being more favorable. As a rule, high altitudes furnish this, but a cold climate is not suited to laryngeal and nervous cases, these doing better in warm climates. Unless the case be one of general miliary tuberculosis, the patient should be sent away as soon as possible.

General Medicinal Treatment.—The drugs that have been found generally useful and have received the sanction of the profession are those that have an influence over general nutrition, that increase the normal physiologic resistance, and render the tissues less liable to invasion.

Such drugs as creasote and its derivatives, benzosol, terebene, guaiacol, arsenic, iodid of potassium, cod-liver oil, hypophosphites, and iron are the most valuable.

Treatment of Special Symptoms.—Rest is the most important factor in the treatment of fever, and the use of drugs is unsatisfactory. Sponging with tepid water is of use, and occasionally combinations of quinin, digitalis, and the salicylates give good results. The coal-tar antipyretics act with promptness in reducing the fever, but the debility is markedly increased by the use of these drugs.

Night-sweats.—Atropin is the most valuable drug for the treatment of this condition. Camphoric acid and aromatic sulphuric acid are also sometimes found efficient.

Cough.—Opium in some form is necessary to alleviate cough. Heroin in small doses is a useful agent.

Diarrhea.—If symptoms of diarrhea occur, bismuth in large doses, combined with opium, will be found efficient.

Treatment of Laryngeal Complications.—Local treatment directed to the healing of the ulcers is often found of use.

Hemoptysis.—Absolute rest, withholding of food, ice-bags applied to the chest, and administration of opium are the most valuable methods for combating this symptom when it becomes serious.

Treatment of Tuberculous Peritonitis—Laparotomy is frequently useful in this condition, and the case should come under the care of a surgeon.

SEPTICEMIA.

Definition.—Septicemia is a disease characterized by the presence of bacterial poisons and bacteria in the blood, and by marked constitutional symptoms without suppuration.

Etiology.—The condition, as a rule, results from some local infective area, as general peritonitis, appendicitis, pneumonia, enteric fever, or gonorrhea. Various micro-organisms have been found in the blood, such as the staphylococcus, the typhoid bacillus, the streptococcus and others, the streptococcus being considered to give rise to the most severe symptoms.

Occasionally, the condition arises without a local infection, and may then be called cryptogenic septicemia.

Pathology.—The blood shows marked changes, both during life and after death. As a rule, a marked inflammatory leukocytosis exists. This may vary somewhat. If the infection be very intense or overwhelming, or if the individual resisting power is not marked, a leukocytosis may not be present, and occasionally a hypoleukocytosis may be found. If the resist-

ing power of the individual is pronounced, or if the infecting agent is not severe, a decided leukocytosis results. As a consequence of the infection, the erythrocytes show a pronounced reduction, and the hemoglobin will also be proportionately reduced. It is said that the most marked oligocythemia exists as a result of septicemia, showing that hemolysis is pronounced. After death the organs may show evidences of hyperemia and cloudy swelling; otherwise nothing specific has been found.

Symptoms.—The condition begins abruptly with chilliness or a decided chill, followed by fever, which rises and falls with slight remissions, however, remaining quite high—from 103° F. to 105° F. The pulse is rapid, and gastro-intestinal disturbances are frequent. Anemia soon begins to be pronounced, and there may be slight jaundice. Purpuric symptoms are not unusual in the course of this disease.

Mental disturbances, manifested by delirium, are quite common, and melancholia not infrequently develops. The condition may terminate fatally within twenty-four hours or more, or the disease may run on for weeks and sometimes months.

Less frequently, septicemia develops without a discoverable primary focus, attacking individuals in perfect health, and at the autopsy the primary focus can not be ascertained.

The urine shows the characters common in febrile conditions, and often contains albumin (toxic) with numbers of bacteria.

Diagnosis.—The diagnosis is made with ease if a primary focus can be found. The type of fever, the pulse, and the examination of the blood revealing a leukocytosis and marked anemia usually determine the true nature of the affection; this condition may simulate malaria, but the detection of the plasmodium is all that is necessary to differentiate between them.

If possible, a bacteriologic examination of the blood should be made in order to determine the character of the microorganism that may be present.

Prognosis.—The prognosis is always to be regarded as serious. A marked leukocytosis will either show good resisting power or a severe infection. Streptococcus infection is regarded as more severe than other forms.

Treatment.—Surgical treatment is, as a rule, necessary, especially when the primary focus exists as an abscess or

some other surgical condition. When the condition is purely medical, such as occurs in pneumonia, the focus should be treated. General constitutional measures must be employed: quinin, antipyrin, and antifebrin may control the temperature for a short time. The temperature may also be controlled by sponging. Strychnin and alcohol should be given as systemic tonics. Some cases show remarkable and brilliant results if antistreptococcic serum is used, when the affection is of this character of infection.

PYEMIA.

Definition.—Pyemia is a condition characterized by marked constitutional symptoms and multiple abscess formation in various parts of the body, and by changes in the blood—and necessarily in many other tissues—due to micro-organisms and their toxins present in the blood.

Etiology.—As a rule, an original focus can easily be determined, such as have already been mentioned in septicemia, the condition being especially common in osteomyelitis, otitis media, empyema, appendicitis, and peritonitis.

Pathology.—Multiple abscesses are found in pyemia, scattered widely through the entire organism. A frequent seat is found in the liver, but no part of the body is exempt. These abscesses result from septic emboli (produced by the breaking up of thrombi), these lodging in the smaller blood-vessels, causing septic infarcts. Abscess formation is the result of the septic emboli; it is possible that the phagocytes of the blood are not able to deal with the micro-organisms as they exist in these particles, while in septicemia the micro-organisms exist as individual units, the phagocytes being able to exert their power, thereby preventing suppuration. As in septicemia, the blood shows changes, leukocytosis being marked and destruction of the red cells pronounced.

Symptoms.—The onset is marked by chill and a rise in temperature, followed by sweating. There is loss of appetite, vomiting, and diarrhea. The chill, fever, and sweat usually occur with great regularity each day or every other day.

As the abscess formation develops, symptoms make their appearance, depending upon the locality in which the abscess forms. Intense pain may be noticed in the region of the spleen or liver. Malignant endocarditis, pericarditis, or pleuritis may develop. Embolic abscesses in the brain substance

will reveal definite symptoms, depending upon their location. Emaciation and anemia are marked as the disease progresses. The condition may last from a few days to two months.

Diagnosis.—The direct diagnosis depends upon the original focus of infection, the characteristic fever-curve, and the symptoms resulting from abscess formation. Leukocytosis should again be sought for. This condition is often confounded with malaria. The presence of the plasmodium in the blood easily differentiates the two conditions.

Prognosis.—As a rule, the prognosis is unfavorable.

Treatment.—Surgical treatment is usually necessary. The condition is treated as is septicemia.

ANTHRAX.

Definition.—An acute infectious disease, occurring as a result of accidental inoculation caused by the bacillus of anthrax. The disease is common in animals, particularly sheep and cattle.

Synonyms.—Malignant pustule; charbon; splenic fever or wool-sorter's disease.

Etiology.—This is a wide-spread disease in animals. It is prevalent in Europe, Asia, Australia, and America. The disease is conveyed to man as a result of the handling of wool or hides, rarely by direct inoculation from the bites of insects that have fed on animals that have died of the disease. It is very rare in man.

Certain occupations predispose, such as stable-men, tanners, butchers, shepherds, and wool-sorters.

The exciting cause is the bacillus of anthrax. (For description of the germ see p. 108.)

Pathology.—A malignant pustule exists, the favorite situation being upon the neck, face, or arms. The lymphatics near this acute inflammatory area will show enlargement. The spleen is enlarged and softened.

Symptoms.—The onset of the local infection is marked by itching and uneasiness. In a short time a papule, soon becoming vesicular, develops. Swelling and redness of a dusky hue soon follow, and in about thirty-six hours marked inflammatory induration exists, the neighboring lymphatic glands being swollen. As a rule, the temperature rises as the inflammation extends, but it may occasionally fall and become subnormal.

The constitutional symptoms depend upon the extent of the local inflammation, and resemble septicemia. As the local inflammatory area becomes more pronounced, suppuration ensues. An intestinal form, described as intestinal anthrax, is marked by symptoms of intestinal toxemia, vomiting, diarrhea, rapid pulse, and fever. Delirium may develop.

Diagnosis.—The diagnosis, which is always difficult, depends upon the finding of the bacillus.

Prognosis.—The prognosis is unfavorable. Death not infrequently results within twenty-four hours.

Treatment.—The pustule should be treated surgically, the site of inoculation being destroyed by bichlorid of mercury, carbolic acid, or the actual cautery. Constitutional treatment consists of the use of quinin, iron, strychnin, and alcohol.

ACTINOMYCOSIS.

Definition.—An infectious disease caused by the ray fungus, or streptothrix actinomyces.

Synonyms.—Lumpy jaw; wooden jaw.

Etiology.—The disease is common in cattle and is rare in man. (For description of the parasite see p. 121.)

Pathology.—The streptothrix actinomyces produces a granulation tumor not unlike that produced by the bacillus of tuberculosis. The small mass consists of proliferated connective-tissue cells, most of them being small; some larger epithelioid or giant cells may also be present. The most common seat of development is in the jaw, where it simulates osteosarcoma. Suppuration develops, and the ray fungus may be found in the pus.

The mode of entrance of the germ in man is commonly supposed to be by the alimentary tract, the fungus being swallowed, or lodging in a decayed tooth, and thence by its growth setting up the symptoms of the condition.

Symptoms.—The clinical course of the disease is usually chronic, the symptoms manifesting themselves slowly. Exceptionally, the onset may be abrupt, due to the rapid growth of the fungus. If the disease occurs in the jaw, rapid swelling of the hard and soft tissues may ensue.

The fungus may enter the larynx and gain access to a bronchus, and the characteristic changes develop in the lung, or it may lodge in the intestine and the growth take place there.

The symptoms may resemble an acute infectious disease or

a pyemic process, but this is exceptional, for if pus-formation result, it may be ascribed to an accidental infection "or to the fungus" (Israel).

When the disease occurs in the jaw, invasion of neighboring tissues, accompanied by great swelling with the formation of cicatricial tissue, occurs. Abscess formation soon takes place, and pus exudes, containing yellowish granules that, under the microscope, show the distinctive character of the growth.

If the lesion be developed in a bronchus, bronchitis is set up, which may be putrid in nature, the expectoration containing the fungus. Abscess formation may result in the lung and adjacent tissues. In the intestine the growth may occur with swellings in the lymph nodules, accompanied by diarrhea. There are also rapid emaciation and loss of strength.

Diagnosis.—The chronic character of the disease, the swellings, the changes in the organs, with the appearance of the yellowish granules in the pus and the identification of the fungus under the microscope, render the diagnosis certain. Vegetable fibers in any discharge (especially pus) should be regarded as suspicious.

Prognosis.—If the disease appear externally in bone or in the skin and *remain* local, a cure may occur; in the viscera (lung, liver, brain, etc.) the prognosis is unfavorable.

Treatment.—Surgical interference whenever practicable is important. The use of iodin and iodid of potassium in large doses has produced a cure in some cases. General supportive treatment is necessary.

MILIARY FEVER.

Definition.—An acute infectious disease, occasionally epidemic, characterized by abrupt onset, profuse sweating, a peculiar exanthem, and constitutional symptoms.

Synonyms.—Sweating sickness; Schweissfriessel.

The first occurrence of this disease was in 1486, in the army of Henry VII, after its return from Bosworth field. The disease appeared epidemically, Hirsch having tabulated 194 epidemics until 1874. In 1887 a severe epidemic occurred in France. The disease occasionally makes its appearance in parts of France and small portions of Italy. It has never been found in this country.

Etiology.—Very little is known of the predisposing causes

or of the exciting cause of this disease. Epidemics have occurred in spring and summer, and the disease appears to be more prevalent in damp and low-lying areas. The middle period of life appears to be predisposing. Women are more liable to attacks than men. The disease is not contagious.

Period of Incubation.—The period of incubation is unknown.

Pathology.—There are no characteristic lesions found after death.

Symptoms.—Prodromes usually occur, consisting of headache, malaise, and weakness, the patient being first attacked at night, with profuse sweating, fever, and pain in the epigastric region. The elevation in temperature is moderate,—101° F. to 102° F.,—and the pulse is rapid. There are tender points over the abdomen. After a variable period—usually from three to four days—an eruption appears, which increases the severity of the previous symptoms; it consists of small reddish points of irregularly circular form, varying in diameter from one-half to one line. The eruption runs together and is confluent. A small vesicle soon appears in the center, which may enlarge to the size of a pea. The contents of the vesicle, at first clear, soon become opaque and purulent, and in two or three days crusts form that are soon cast off. The eruption is sometimes found upon the mucous membrane of the nose, mouth, and conjunctiva. As a rule, it first appears upon the neck and chest, thence upon the back and extremities.

The cases differ in severity from the mild to the most severe types. In the gravest cases nausea, vomiting, delirium, and coma may occur, the patient dying in the so-called "typhoid" state.

Convalescence, even from very mild cases, is prolonged.

Diagnosis.—The diagnosis depends upon the occurrence of an epidemic, with appearance of sweating and the characteristic eruption. It must be carefully differentiated from malaria, variola, measles, and rheumatic fever.

Prognosis.—The prognosis varies in different epidemics. Occasionally the disease has been very mild, and in some epidemics the mortality has reached from 50% to 80%. When death occurs, it usually takes place during the sweating stage.

Treatment.—The treatment is purely symptomatic. Specific treatment does not exist. Quinin in moderate doses seems to exert some influence. In convalescence tonics are required.

MOUNTAIN FEVER.

A disease has been described, occurring in mountainous regions, especially in the Rocky Mountains of the United States, with symptoms of extreme palpitation of the heart upon exertion, headache, giddiness, nausea, vomiting, marked dyspnea, and rapid pulse. These symptoms have been ascribed to the rarefied air. This condition must not be confounded with the disease called mountain fever.

The group of cases to which the term "mountain fever" has been applied present symptoms of an irregular continued fever. The duration of the attack is from two to four weeks or longer, and the temperature may vary between 101° F. and 104° F.

It is most probable that this is a form of enteric fever or croupous pneumonia, as lesions of both these diseases have been found in cases dying from so-called "mountain fever."

MILK-SICKNESS.

Definition.—An acute disease particularly affecting cattle, occurring in man probably as a result of eating the flesh or drinking the milk of an animal so affected, characterized by great weakness, vomiting, constipation, twitching of the muscles, and other constitutional symptoms.

Synonyms.—Trembles; puking fever; slows.

Etiology.—Three theories have been advanced to explain the causation of the disease: (1) That the poisonous principle is furnished by a variety of rhus; (2) That it is due to poison contained in the drinking-water of cattle; (3) That it is due to a specific germ. It must be admitted, even at this day, that the etiology is still unsettled.

Pathology.—Few opportunities have occurred for the performance of autopsy. The pathologic findings have been principally studied in cattle. Changes have been found in the cerebrospinal system, consisting of congestion and effusions of blood into the membranes. The liver and spleen are soft, and often enlarged and engorged with blood. The occurrence of meningitis shows a possible analogy to cerebrospinal fever.

Symptoms.—The symptoms occur abruptly, although the onset of the disease may be insidious. The principal symptoms consist of the appearance of tremor, with marked mus-

cular weakness, vomiting, and fetor of the breath. The tongue is coated, dry, swollen, and fissured. Vomiting is a frequent symptom. The bowels are obstinately constipated, the pulse is full at first, but soon becomes small and rapid. The temperature may be slightly elevated, but in cases coming on abruptly the temperature may be subnormal.

If death occurs, it is often preceded by symptoms of hiccup and delirium, finally passing into coma.

Diagnosis.—It is most probable that this is a disease closely allied to cerebrospinal fever.

Prognosis.—Favorable, depending upon the degree of toxemia.

Treatment.—The treatment should be the same as in cerebrospinal fever.

LEPROSY.

Definition.—A chronic specific disease, characterized by cutaneous pigmentary changes and by the formation of neoplasms in the skin, mucous membranes and nerves, which give rise to alterations in sensation, to ulceration, and to progressive deformity.

Synonym.—Elephantiasis græcorum.

The disease is known in various languages as leprosy.

There is abundant proof that leprosy is an ancient disease; a description of the disease corresponding to leprosy existed, according to Brugsch, at 4600 B. C., long antecedent to the Mosaic exodus.

The disease still exists in certain parts of the world, and has occurred endemically in many places in the tropics, in the temperate zones, and even in the Arctic circle.

Etiology.—Predisposing Causes.—The hereditary transmission of leprosy is still open to doubt. Many reliable observers claim that it is hereditary, and, on the other hand, this is doubted by equally good authority. Poverty and improper hygiene have an undoubted effect on the disease. Improvement in the mode of living, proper food, and cleanliness have driven the disease out of the populous portions of Europe. No race is exempt, and the disease occurs in all climates.

Exciting Cause.—The exciting cause is the bacillus lepræ. (For description of the germ see p. 107.) Inoculation of the human subject with leprous tubercles has been successful. The disease is endemic in certain parts of the world.

Pathology.—The postmortem changes show characteristic granulation neoplasms, the new growth being mostly made up of round cells, the bacilli being contained in the mass. The skin, the mucous membranes, nerves, lymphatic glands, and various organs of the body show these changes.

Period of Incubation.—This may extend through many years, and the period has not been definitely determined; so much is, however, certain, that the period of incubation must be reckoned by years rather than by months.

Varieties.—The disease has been divided into various varieties: (1) *The nodular or tubercular form*, in which the skin is primarily and chiefly affected; (2) *the smooth*, in which the nerves are primarily affected; (3) *the mixed form*, in which the lesions occur both in the skin and in the nerves. These varieties are not always distinct, one form readily merging into another.

Symptomatology.—The Nodular or Tubercular Variety.—There are usually prodromes, which consist of an irregular rise in temperature, associated with lassitude, drowsiness, gastro-intestinal symptoms, headache, backache, sometimes bleeding at the nose, and, occasionally, profuse perspiration, associated with rigors. These may be slight or absent altogether.

The first positive evidence of the disease consists in the leprous eruption, which appears as an irregular, shiny, erythematous patch of a reddish or copper tint, slightly raised above the surrounding skin, with infiltrations of the derma, accompanied by more or less hyperesthesia. The parts first affected are generally the lobes of the ears, the alæ of the nose, the forehead, the eyebrows, the roots of the nose, and the lips; in other words, the face is *always* primarily affected. Later, the eruption appears upon the extremities and the trunk. It may persist, or it may partially or even completely disappear for the time, but it invariably returns. Upon its return the well-marked leprous nodules show themselves as small papules that gradually enlarge, so that in course of time they may attain the size of a pigeon's egg. They appear upon the site of the former eruption. Small blood-vessels may sometimes be seen over the nodules. Occasionally, the entire body is invaded by this process. The hairy scalp, however, is rarely affected. This is known as the second stage of the disease. The nodules may now remain stationary

or may increase in size, in rare cases disappearing altogether. The face becomes characteristic, the region occupied by the eyebrows becomes prominent, and the forehead greatly thickened. A somber expression of the countenance is noticed.

In a majority of cases the growth increases rapidly, fresh crops appearing from time to time, each being accompanied by a rise in temperature—from 100° F. to 103° F. The lymphatic glands become prominent and painful, and the hands and feet are covered by the new growth, the nails dropping off.

After this period has lasted a variable time the stage of ulceration sets in. In favorable cases some of the ulcers may heal, the patient living for some time; in others the ulceration spreads by irregular tracts, bleeding taking place in the ulcerated nodules, especially upon the hands and feet. This condition may last for months or years, the patient dying from asthenia or visceral complications, such as renal disease or tuberculosis.

The Smooth Variety (known as the Anesthetic or Atrophic Form).—The prodromes consist of alterations in sensation with trophic changes. These may be ill defined and come on slowly. There may be chilliness and general depression. Pain along the nerve-trunks is apt to occur, or if absent, may give place to numbness and tingling in the hands and feet. The sphincters may become affected. In some cases the eruption is the first symptom to appear; it may be erythematous or pigmented. The spots are usually light copper or brown, or in the black races dirty yellow in appearance. The eruption commonly appears first upon the shoulders, back, loins, knees, elbows, and exceptionally upon the face. The face, however, may escape altogether. The eruption may follow the course of the musculospiral nerves. Fresh patches continue to appear, although the patient's health may not be otherwise affected. Usually, however, changes in sensation begin to appear, particularly anesthesia in the course of the ulnar nerves, more commonly in the left hand than in the right.

New areas of the eruption begin to appear, although the original spots remain unchanged for several years. The hair falls out, and there is destruction of the sweat-glands. Perspiration is absent in this variety. The superficial nerve-trunks may be felt enlarged beneath the skin.

After an indefinite period the eruption ceases to be active,

and there is no further deposit in the nerve-trunks. The disease may now remain stationary for a number of years. In the majority of cases, however, a continuous destruction of nerve tissues results, due to increasing leprous growth in the nerve-trunks. This gives rise to contractures.

Arthropathies take place, resembling Charcot's joints. The bones may ulcerate, due to a lack of nerve supply. Ulcerations are common upon the fingers and toes; the nails split and break. Eye affections in this form are due to lesions of the cranial nerves.

Mixed Leprosy.—The symptoms in this form show a combination of the two just described.

Diagnosis.—In the fully developed disease no difficulty is encountered in making a correct diagnosis.

In countries where the disease exists, all new growths should be looked upon with suspicion, especially if combined with altered sensation. The finding of the bacillus is positive evidence.

Prognosis.—The prognosis is grave, the disease being incurable.

Treatment.—Much may be done to prolong life and to mitigate suffering. Hygienic treatment is of the greatest importance—fresh air, well-ventilated and dry dwellings, and strict cleanliness are important. Extremes of heat and cold should be avoided. A good nourishing diet, consisting largely of fresh vegetables, should be given; warm clothing is essential, as the patients are very susceptible to cold and liable to disease of the kidneys. There is no specific treatment.

The vegetable oils are most frequently employed, and oleum gynocardiæ or chaulmoogra oil appears to be the best to increase perspiration, improve appetite, lessen anesthesia, and diminish pain.

Arsenic is useful in some cases, and should be given to the point of tolerance. Ichthyol and resorcin have been advised by Unna. Thyroid extract has been efficacious in some cases.

When gangrene occurs, amputation of the part may be necessary. Operations may be performed upon the leper without hesitation. Dead bone should always be removed. Nodules giving rise to obstruction should be excised. Tracheotomy prolongs life when the larynx becomes invaded and dyspnea is threatened.

DYSENTERY.

Definition.—Acute dysentery is a disease, principally of the large bowel, with or without fever, characterized by diarrhea, which is often bloody in character, with tormina and tenesmus.

The term "dysentery" should be made to include all forms of alvine flux, either mild or severe, in which tormina and tenesmus are prominent symptoms (Trousseau).

Synonyms.—Flux; bloody flux; Ruhr (German).

Varieties.—On account of the diverse symptomatology, it has been necessary to arrange this disease into four principal varieties: (1) *acute catarrhal dysentery;* (2) *diphtheric dysentery;* (3) *amebic or tropic dysentery;* (4) *secondary dysentery.*

Etiology.—Climate is of great importance, as this is particularly a disease of tropic and subtropic regions, the malady prevailing to a greater extent during the hotter months. All ages are susceptible, and if enterocolitis be considered dysenteric in nature, it occurs as frequently in children as in adults. The male sex is more liable to the affection, on account of greater exposure, such as in camp life, armies, fleets, and prisons.

This disease has been the most frequent cause of death in times of war, and Osler remarks that "it has been more fatal than shot and shell." Change of weather is an important factor, as sudden falls in temperature at night or exposure to heavy rain and dew, as frequently happens when camping upon the ground. Bad hygiene is probably one of the foremost predisposing causes. Overcrowding, irregular taking of food, bad water, especially if it be polluted, forced marches, and all conditions tending to lower the vital resistance may give rise to dysentery.

Bacteriology.—Various forms of bacteria, but no constant one, have been found associated with dysentery. It has been claimed by many that the bacillus coli communis (see p. 110) takes upon itself special pathogenic properties under certain conditions and produces this disease. It is probably due to the combination of various forms of bacteria with the absorption of their ptomains. The amœba dysenterica has frequently been found associated with tropic dysentery.

ACUTE CATARRHAL DYSENTERY.

Pathology.—This form most frequently terminates in recovery, and postmortem studies have mostly been made where an intercurrent affection has produced death.

The colon is most often involved, and the process may extend as far as the rectum. Occasionally, the ileum and parts of the small intestine may also show the lesion. The mucous membrane is congested, swollen, and red, covered with a thick, opaque, yellowish or brownish mucus. The submucosa also is swollen and injected, pus being often present. The lymph nodes may be found enlarged. Ulceration may occur if the case be protracted.

Symptoms.—The onset marked by chill and fever is extremely rare, the attack beginning as an ordinary acute intestinal catarrh, which, after a few hours or days, takes on the additional symptoms of dysentery. These consist of pain and griping, commonly in the umbilical region and along the colon, accompanied by a dull pain in the loins.

The stools at first may be either copious or scanty, soon becoming free from fecal material. There is frequent desire to have a passage from the bowel, accompanied by severe pain. This condition has been called *tormina*. However, spasms frequently occur at this time, and nothing but a slight amount of bloody mucus or pus is passed. This is known as *tenesmus*. The stools may consist entirely of blood mixed with mucus, and the association of pain with spasm may take place from every few minutes to half an hour or longer; indeed, it may be almost continuous. After the first few days there are very slight, if any, constitutional disturbances, even if the local symptoms be quite severe. Later, there are loss of appetite, coated tongue, nausea, sometimes vomiting, with slight evening fever—100° F. to 101° F. This condition may continue for some time, the symptoms gradually ameliorating, or the symptoms may increase in severity and the disease pass into the diphtheric variety, or they may persist for a long time and become chronic.

Prognosis.—In temperate climates under ordinary conditions the prognosis is almost invariably favorable.

DIPHTHERIC DYSENTERY.

Pathology.—The colon in the majority of cases is principally affected, and the diphtheric process most frequently follows the acute catarrhal one. The diphtheric process varies

greatly in extent as to firmness and tenacity of the exudate. The exudation is sometimes limited to the colon and the rectum, but occasionally the rectum alone is affected. It may extend into the small intestine. In some cases the exudation is quite superficial, filling the follicles of Lieberkühn. Most frequently, however, the submucosa is involved. Under the microscope the exudation has the croupous character. The loss of substance in the separation of the exudate is sufficient in some cases to cause ulceration. These ulcers vary in size from a slight abrasion to a deep excavation, which may invade the muscular coat of the bowel. Death may occur before the sloughing is completed. Perforation of the serous coat may occur, death being due to peritonitis. When healing takes place, the cicatrix is more or less puckered, and in rare instances stricture of the bowel results from this cause.

Symptoms.—The disease may commence as an ordinary acute catarrhal dysentery, or it may begin abruptly, following an ordinary diarrhea, or the dysenteric symptoms may occur at once, without any preceding stages. It sometimes results from other acute or chronic diseases, and shows itself by the passage of blood and mucus. The patient becomes emaciated and weak, with symptoms of collapse, cold surfaces and extremities, and marked increase in the cardiac action. The tongue is dry and fissured, and anorexia and thirst are prominent. The abdomen is tender to the touch and tympanitic. The skin may be slightly jaundiced, and the eyes sunken. With the occurrence of these symptoms a change in the character of the stool takes place. At first it consists of stringy mucus, more or less mingled with blood and pus. Yellowish or reddish masses, of various sizes, of the diphtheric exudate, which under the microscope are seen to be necrosed tissue, may be found. The passage of this diphtheric exudate is characteristic. There are great tormina and tenesmus. The urine is decreased in amount and often contains albumin. The disease may last from one to four weeks from the beginning of the dysenteric symptoms. Just prior to death the stools become brownish or black and are very offensive, and contain large amounts of necrosed tissue.

The onset of the disease is sometimes accompanied with a chill, followed by fever, which may run to 103° F. or 104° F., with marked abdominal symptoms. Nervous symptoms are rare. The patient rapidly passes into the typhoid condition ; the pulse is rapid—120 to 140 ; sordes collect upon the

teeth, and the patient rapidly passes into collapse with the characteristic stool just described.

When diphtheric dysentery occurs in the progress of a pre-existing flux, it may appear suddenly and rapidly prove fatal; this is frequently the case after exposure to cold, indiscretion in diet, and even sometimes without assignable cause. The characteristic stools develop, and fatal collapse is rapid.

In chronic cases the stools show the diphtheric character later in the progress of the disease. The symptoms are not severe, and the case may end in recovery in from two to four months, or pass into chronic dysentery, in which the duration is indefinite. All these phenomena may be modified in different cases.

AMEBIC (OR TROPIC) DYSENTERY.

Pathology.—The large bowel is most frequently implicated, as the ameba has here the most favorable condition for its development. The small intestine may become infected; in such cases the ameba passes from the large into the small bowel. The characteristic change consists in the great thickening of the intestine, which is usually more marked in the submucous coat, and may be confined to this part. The ameba is found in the discharges and is also present in the coats of the bowel. (For description of the ameba see p. 761.)

Abscesses of the liver frequently result as a consequence of this condition. They are generally large and single, but two or three may occur in the same case.

The infection is probably carried to the liver by the portal circulation, but this is disputed by some authorities. Abscess of the lung sometimes results.

Symptoms.—Several varieties have been described, such as the *mild*, the *grave*, and the *chronic*, with masked symptoms. The onset is variable, with intermissions and exacerbations. It may be abrupt or gradual, the patient being suddenly attacked by severe pain in the abdomen. Diarrhea and nausea and vomiting are sometimes present, mostly without fever. The stools are frequent and watery, and blood, which may be present from the first, subsequently appears combined with mucus. In some cases abdominal symptoms may be absent altogether, and all the signs point to a hepatic or pulmonary abscess. In ordinary cases the tongue is pale and flabby; the abdomen is usually normal in appearance; the expression is dull; nervous

symptoms are absent. There is no fever; the pulse may be from 70 to 90 a minute. Anorexia is present.

The diagnosis depends upon the finding of the ameba in the discharges.

SECONDARY DYSENTERY.

Pathology.—The lesion in this variety is generally the same as described under the head of diphtheric colitis.

Symptoms.—This is usually the terminal event in various chronic and acute diseases, such as cardiac affections, Bright's disease, smallpox, septicemia, and tuberculosis. The stools have the characteristic appearance of dysentery in general—blood and mucus. There may be from three to a dozen movements in twenty-four hours, and death may occur without symptoms of tormina. Peritonitis is not infrequent.

Complications.—The most usual complications are anemia, peritonitis, with an intestinal perforation, hepatic and pulmonary abscesses, pneumonia, tuberculosis, and malarial fever.

Sequels.—Among the most important sequels of dysentery are fistulas from abscesses and hemorrhoids, with prolapse of the anus.

Diagnosis.—The diagnosis depends upon the careful examination of the stool. In the acute catarrhal variety there are blood and mucus; in the diphtheric variety, the appearance of necrosed tissue; in the amebic variety, the presence of the ameba in the evacuations; in chronic dysentery, the association with some other important disease.

Prognosis.—In the acute catarrhal variety the prognosis is favorable. In the diphtheric variety, especially that occurring in camp life, the mortality is often enormous: it may average from 50% to 80%. The prognosis in children depends upon the age and the constitutional condition of the patient. Persistent high fever, vomiting, rapid wasting, and severe nervous symptoms are of unfavorable prognostic omen. The prognosis in the amebic form is always uncertain.

Treatment.—Prophylaxis should consist in general hygiene, particular attention being paid to the drinking-water. Great care should be taken that there is no fecal contamination of the water-supply. It is considered good practice to give, early, a mild cathartic. The drugs that have given the best satisfaction are ipecac, opium, and the bismuth salts. Enteroclysis is of value in the management of dysentery.

SYPHILIS.

Definition.—Syphilis is an infectious disease, characterized by a primary lesion called a chancre, secondary manifestations, and tertiary lesions. It may be either congenital or acquired.

Etiology.—The disease in the majority of cases is transmitted by sexual intercourse. It may, however, be conveyed in other ways. Infection may take place in the practice of the physician or surgeon, he becoming accidentally inoculated by the examination of a syphilitic person.

Vaccinal syphilis has occurred, but since the almost constant use of bovine virus this accident is rare.

Syphilis has been acquired from the use of tableware, instruments, or articles used by a syphilitic person. It has also been conveyed by kissing.

Hereditary Transmission.—It occurs most commonly from the father, the mother being healthy (sperm inheritance).

Maternal Transmission.—(Germ Inheritance.)—It is a well-known fact that a woman who has borne a syphilitic child may be immune. This has been stated by Colles, and is known as Colles' law: "That a child born of a mother who is without obvious venereal symptoms, and which, without being exposed to any infection subsequent to its birth, shows this disease when a few weeks old, this child will infect the most healthy nurse, whether she suckle it or merely handle and dress it; and yet this child is never known to infect its own mother, even though she suckle it while it has venereal ulcers of the lips and tongue." A mother with acquired syphilis may have infected children and the father not be affected. In the greatest number of cases both parents are affected, the one having affected the other, in which case the fetal infection is greatly increased.

If the mother be infected after conception, the child may be born syphilitic, although not necessarily so. This may occur through the placenta.

Pathology.—Exciting Cause.—The specific cause has not been definitely determined, but the organism that at the present time has the best claim to recognition is the bacillus of Lustgarten.

Chancre.—This lesion consists of a number of round cells infiltrated into the tissue (perhaps the bacillus is intermingled in this mass). The blood-vessels in its locality undergo thick-

ening by connective-tissue formation. The tissue elements of the skin and subcutaneous tissues are usually surrounded by infiltrating liquid, and the latter may cause the exfoliation of the superficial epidermis and thus lead to the development of the ulcer so commonly seen.

The induration of the initial lesion is due to the sclerosis of the vessels, newly formed connective tissue, infiltration of the embryonic cells, and liquid infiltration.

Secondary Manifestations.—Secondary manifestations are numerous and very variable. The most important of these are the skin eruptions and the involvement of the mucous membranes and lymphatic enlargements.

Tertiary Lesions.—The gumma, or tertiary lesion, strictly speaking, is the typical syphilitic manifestation. It consists of a central caseous area surrounded by a number of small round cells.

Occasionally, some of these cells may be somewhat enlarged and are called epithelioid cells. Giant cells may also be found. The size of the gumma varies from that of a millet seed to the size of a walnut, and at times is even larger.

The arteries near this lesion commonly show sclerotic changes. After a prolonged existence the gumma, if situated near a free surface, may discharge its caseous area and form an ulcer. Healing takes place by the formation of connective tissue leaving a cicatrix, which is frequently stellate.

Site.—The liver, kidneys, spleen, bones (especially the tibia), brain, and nervous system are favored localities.

ACQUIRED SYPHILIS.

Symptoms.—The symptoms are usually divided into three stages: *The primary, secondary, and tertiary stages.*

Primary Stage.—The period of incubation varies from ten to ninety days, the average being about three weeks, and the first symptom noted is the primary sore. This most generally appears about a month after inoculation. It consists of a red papule that enlarges, leaving a small ulcer. The tissue surrounding it becomes indurated, so that it has a cartilaginous or gristly consistence, and is often called the hard or indurated chancre (Hunterian chancre). It varies in size and, when very small, may be readily overlooked, especially if it occurs within the urethra. The lymph-glands in the neighborhood of the chancre enlarge and become hard. Suppuration rarely occurs. There is usually no fever or decided

impairment of health. The most common situation of the chancre is upon the external genitalia. Lymphatic enlargement soon appears.

Secondary Stage.—This makes its appearance within from one to three months, the average being about six weeks after the appearance of the chancre. There is fever, which is very variable, being either intense or the reverse, although most commonly it is mild. The fever often has a decidedly remittent character; on the other hand, it may be intermittent. It may occur later in the disease, and be one of the tertiary symptoms.

Anemia often of the chlorotic type is an important symptom. The first symptom noticed in the secondary stage is the cutaneous lesion, the most common being the macular syphilid or syphilitic roseola. This occurs on the abdomen, chest, and extremities; the face may escape. It has a reddish-brown or coppery appearance. Other eruptions also occur: the papular, pustular, and the squamous syphilid. Mucous patches develop. They are found upon the perineum, the groins, the axillæ, between the toes, and at the angles of the mouth, and especially upon the mucous membranes. They are flat, warty outgrowths, and their surface is covered with a grayish secretion. The hair falls out. This may occur either in patches or there may be a general thinning, especially in the temporal regions. The nails may also become affected. Lesions of the mucous surface are very common. The tongue, the pharynx, and the tonsils may show characteristic syphilitic ulcers. Syphilitic warts or condylomata may occur in the genital regions and the anus. Other parts of the body may also be affected. Eye symptoms are very common, ear affections may occur, and general lymphatic enlargement is present.

Tertiary Stage.—It is extremely difficult to say when the secondary manifestations cease and the tertiary begin. The special affections of the tertiary stage are gummatous growths in the viscera, certain skin eruptions, and rarely amyloid disease.

The gumma may develop in the skin, subcutaneous tissues, muscles, bones, or internal organs.

It is characteristic of the late skin manifestations of syphilis that there is a liability to ulceration. The most characteristic of these is rupia, which consists of dry, stratified crusts that cover the ulcer and that, upon healing, leave a permanent scar.

Among the important symptoms of tertiary syphilis are bone-pains, which are especially marked at night.

CONGENITAL SYPHILIS.

The symptoms in this variety are similar to those already described, with the exception that the primary chancre does not exist. An apparently healthy-looking child may be born without showing any symptoms whatever of syphilis for a month or two.

Pathology.—Among the lesions observed are sclerotic changes in the liver, lungs, spleen, pancreas, bones, and other organs. Skin manifestations are also encountered. The upper incisors are notched on the cutting surface, and are known as Hutchinson's teeth.

Symptoms.—At birth the child is usually feeble, and the skin eruption may be present, showing itself particularly about the wrists and ankles and upon the hands and feet. *The child snuffles.* The mouth is fissured and the lips are ulcerated. The liver and spleen are enlarged, and bone symptoms may be prominent. The epiphyses may be entirely separated. In such instances death may take place rapidly. Again, the child may thrive, become plump, and show no symptoms of syphilis whatsoever until some time between the second and third months, when nasal catarrh develops, interfering with respiration, and the characteristic snuffles show themselves. The nasal discharge may be seropurulent or bloody. Necrosis of bone takes place rapidly, and the nose undergoes characteristic deformity. Cutaneous lesions may develop about the same time. They are those that have already been described.

Disease of the nails is frequent (syphilitic onychia). Enlargement of the glands is not characteristic.

The spleen is usually enlarged. The enlargement of the liver, however, is not so prominent.

Hemorrhages are liable to occur; the bleeding may be subcutaneous, from the mucous surfaces, or from the umbilicus. The child does not thrive, but soon becomes cachectic in appearance, although this is not invariably the case.

The teeth are deformed, as Hutchinson has pointed out. Eye affection is very common among the later manifestations, as is also middle-ear disease. Bone lesions do not occur until later—from the sixth year on. Gummata may occur in the brain, liver, kidneys, and other organs.

VISCERAL SYPHILIS.

Gummata may occur in the brain and spinal cord, and their meninges, in the lung, the liver, the digestive tract, the heart, the kidneys, and the testicles.

Diagnosis.—The diagnosis is easy. This disease can scarcely be confounded with any other. In doubtful cases careful inspection should be made of the throat and skin for old lesions. The bones should be examined for nodes, and the scar of the primary sore should be looked for. In doubtful cases the therapeutic test may be resorted to.

Prognosis.—In cases that are treated early the prognosis is favorable.

Treatment.—Treatment should be begun as soon as signs of the secondary manifestations appear. The specific for syphilis is mercury and the iodid of potassium. Tonics are useful in the tertiary stage, and also in cachectic individuals.

GLANDERS.

Definition.—Glanders is an infectious disease due to a specific cause, occurring in animals, and exceptionally in man. It is characterized by enlargement of the lymphatic glands, especially the submaxillary and parotid chains, with symptoms referable to the mucous membranes, particularly of the respiratory tract.

Synonyms.—Farcy; equinæ; Rotzkrankheit (German).

Etiology.—All occupations that bring man into contact with domestic animals, especially the horse, predispose. For this reason the male sex is more frequently affected. Veterinarians, hostlers, coachmen, etc., are particularly liable. The exciting cause is the bacillus Mallei, discovered by Löffler and Schütz in 1882. (For description of the germ see p. 107.)

Pathology.—The cadaver presents the appearance common to pyemia; the surface is covered with pustules, abscesses, and ulcers, particularly upon the face and extremities. It is peculiar of these excoriations that blood is present in them. In this respect they differ markedly from pyemic abscesses. Extensive erosions, which have been caused by cicatricial contraction, and ulceration of the nasal tissues with necrosis of the nasal bones, are characteristic. Nodules are found in the respiratory tract, lungs, brain, liver, and spleen. These nodules are hard and firm, varying in size from small visable points to

a pea or walnut. On section, they are grayish white, having a yellowish-white center. This center is soft and caseous; around this may be found epithelioid cells, leukocytes, and small round cells. Sclerotic changes are sometimes found around the nodules. The lymph-glands near the affected area may be enlarged and infiltrated. Erysipelas of the skin is common. Serous and seropurulent effusions occur in the joints and serous cavities. Occasionally, the effusions are hemorrhagic.

Period of Incubation.—In artificial inoculation the period of incubation is a short one, being about twenty-four hours. In other cases it varies from three to five days.

Symptoms.—The first symptoms are usually redness and swelling, which appear at the point of inoculation. Pain, with swelling of the neighboring lymphatics, occurs coincidently. Constitutional symptoms may precede this, consisting of chilly sensations, headache, prostration, and fever. The joints become painful and are red and swollen. This is characteristic of farcy. Red, hard nodules, varying in size from that of a pea to a walnut, soon appear upon the skin, and in some cases may resemble the eruption of smallpox. They soon break down, however, being rapidly formed, and, upon bursting, the pus is noticed to be very thick and fetid. The nodules may increase in size so that actual tumors are formed, which are known as *farcy buds;* or ulceration, destroying the tissues to such an extent that the bone may be exposed. Occasionally, this process is extremely rapid, and may vary from a day to three or four weeks.

Symptoms relative to the nose are less frequent in man than in the horse. Occasionally, there is ozena, and farcy buds may appear upon the nose. The whole respiratory tract thus becomes affected. Cough is prominent, accompanied by great dyspnea and profuse fetid expectoration.

Fever may or may not be present. It may be very high, —106° F.,—with distinct variations such as occur in pyemia. A chronic variety sometimes shows itself with the same symptoms as those just enumerated, excepting that the process is much slower and may last through years. When farcy buds appear in the internal organs, symptoms referable to these viscera occur.

Diagnosis.—The diagnosis depends upon the exposure, the marked ozena, the nodular eruption, with formation of pus, and ulceration. The bacillus Mallei is found in the pus

from the discharges, and in doubtful cases inoculation experiments should be performed.

Mallein, which bears a resemblance to tuberculin in tuberculosis, is used in veterinary practice for diagnosis in doubtful cases. A reaction always occurs if farcy is present, except in cases accompanied by fever.

Prognosis.—The disease is invariably fatal in man, except in rare instances in the chronic variety.

Treatment.—Prophylaxis is most important. When abscesses and ulcers form, strict antiseptic treatment is necessary. Tonics should be administered as a supportive treatment.

FOOT-AND-MOUTH DISEASE.

Definition.—A mild infectious disease occurring in animals and in man, particularly through infected milk. It is characterized by a slight fever, aphthous stomatitis, benign course, and short duration.

Pathology.—Cases are rarely fatal; ulcers form upon the mucous membrane, particularly of the cheek, tongue, and upon the hands.

Period of Incubation.—The period of incubation varies from three to five days.

Symptoms.—The disease begins with a chill or chilliness, anorexia, and high fever. The first symptoms noticed may be the appearance of vesicles upon the inner surfaces of the lips and tongue, having an inflammatory base; coincidently, difficulty in chewing and speaking occur. This is followed by a vesicular eruption upon the fingers and hands, with perhaps slight gastro-intestinal disturbances.

The vesicles are at first transparent, but upon increasing in size become purulent and may burst. The duration of the disease is from five to eight days, the majority of cases terminating in recovery.

Diagnosis.—The diagnosis consists in the eruption of vesicles upon the buccal mucous membrane and hands, with fever. The knowledge of the disease existing among animals in the district is of importance.

Prognosis.—The prognosis is almost invariably favorable. Few fatal cases have been reported, these having occurred in strumous and delicate children.

Treatment.—Prophylaxis consists in boiling the milk. The disease should be treated by mild alkaline washes and a laxa-

tive dose of calomel at the onset; if pain is prominent, the administration of small doses of Dover's powder or other form of opium is advisable.

HYDROPHOBIA.

Definition.—A disease of animals, especially of the dog, also occurring in man through inoculation, most often due to a bite. It is characterized by severe nervous symptoms, consisting of spasm, with constitutional phenomena, and is very fatal.

Synonym.—Rabies.

Etiology.—Probably due to a specific germ not yet isolated, and always occurring in man through inoculation. The poison is contained in the saliva. The disease is probably due to an organism that multiplies in the tissues and produces a toxin that appears to act specially upon the central nervous system.

Period of Incubation.—The period of incubation varies greatly. The disease may be latent, and no symptoms may occur for from ten to twelve months. In the majority of cases, however, symptoms will manifest themselves in from three to six weeks after the bite.

Pathology.—There is congestion of the spinal cord and brain, and some exudation into the perivascular tissue. Hemorrhages also take place in the cerebrospinal system. The mucous membranes of the respiratory system and gastro-intestinal tract and kidneys may be congested. The toxin is not found in the internal organs, such as the liver, spleen, and kidneys, but is quite abundant in the brain, the spinal cord, and the nerves.

Symptoms.—At the end of the period of incubation the wound becomes painful, there is itching and tingling, with a sensation of heat, which may be accompanied by sharp pain following the course of the nerves. Occasionally, the wound may open afresh and an unhealthy purulent discharge make its appearance. Small vesicles may appear around the wound, which may ulcerate.

In the early stages the patient is feverish and very thirsty, depressed and irritable. The muscles of the face are drawn, and there is a marked pallor and a peculiar look about the eyes. In talking, the speech may be interrupted by a sighing inspiration. The patient is unwilling to talk about the wound or bite

that may have caused his illness. Sleep is disturbed. There is much thirst, and difficulty in swallowing, loss of appetite, nausea, and epigastric pain. The pulse becomes rapid, and the respirations are hurried and shallow.

Upon the second or third day great nervous excitement appears. Delirium may occur. The pallor becomes more marked, the eyes are bright, and the mucous membrane of the mouth and fauces becomes congested. Upon these mucous surfaces an accumulation of thick, tenacious mucus will be noticed, which the patient tries to expel with a coughing sound, not unlike the bark of a dog.

Thirst is prominent, but there is great difficulty in swallowing, especially of fluids. As attempts at deglutition are made, violent spasmodic contractions of the muscles occur. This may be followed by tetanic convulsions, with marked opisthotonos and temporary cessation of respiration. The reflexes are increased, especially the tendon reflex. The symptoms may abate, only to recur again at an attempt to swallow, especially fluids. Slight causes may bring on the spasm, such as a sharp sound, a bright light, a breath of air, or the mere sight of water. The delirium is intermittent, but the patient in this condition may be brought to consciousness by the attendant.

At first there is increased sexual desire, and later this symptom may become even more marked. The urine may contain sugar, albumin, and blood. The repeated attacks of spasm greatly exhaust the patient, and if the disease be protracted, wasting occurs.

Diagnosis.—The diagnosis depends upon the knowledge of a bite from a rabid animal. The appearance upon the second or third day of the peculiar spasm, with the bark, delirium, the occurrence of albumin and sugar in the urine, and the marked tetanic spasms.

Prognosis.—The percentage of death varies from 5% to 50%. Since the introduction of the Pasteur treatment the mortality has been greatly reduced. When the symptoms are well developed, the patient almost invariably succumbs in from four to five days.

Treatment.—The treatment consists in giving antispasmodics, such as chloral, bromid of potassium, cannabis Indica, and curare. Recently, most cases have been treated by the antirabic serum of the Pasteur Institutes.

TETANUS.

Definition.—An infectious disease, characterized by spasm, particularly of the muscles of the jaw, and other symptoms, relating to the nervous system.

Synonym.—Lockjaw.

Etiology.—The disease occurs in either sex and at any age. It may result from a wound in any part of the body and sometimes without apparent trauma. It more frequently follows wounds of the extremities than wounds of the trunk. It occurs most often from injuries that are exposed to contact with earth or filth. The slightest causes may give rise to tetanus, such as a scratch, the extraction of a tooth, or the plugging of a nostril for epistaxis. It is more frequent in man than in woman, but it is said to be less fatal in the female.

Exciting Cause.—The exciting cause is the bacillus of tetanus, described by Kitasato. (For description of bacillus see p. 113.)

Pathology.—Postmortem appearances are not characteristic. Congestion of the spinal membranes and nerves is usually found. The bacillus occurs in the neighborhood of the wound and not in the internal organs, as the toxins are responsible for the manifestation of the disease.

Period of Incubation.—The period of incubation is about two weeks.

Symptoms.—The prominent symptoms are the occurrence and recurrence at varying intervals of tonic spasms of greater or lesser intensity in the voluntary muscles. The spasm may relax somewhat in sleep, but not entirely. It may commence in the neighborhood of the wound and spread over the remainder of the body.

In man the spasm is usually first noticed in the neck, beginning like an ordinary rheumatic torticollis. This is soon followed by spasm of the muscles of the jaw, called trismus, with inability to open the mouth. The tongue may protrude between the teeth, and may be bitten by the violent and sudden closure of the mouth. The facial muscles show a strained appearance, which gives rise to the peculiar grin called the "risus sardonicus."

The muscles of the abdomen become firm and contracted. There may be pain in the precordia, extending through to the back. The spasm affects the voluntary muscles; the fingers, however, are rarely implicated. The pain is frequently not

severe; it may, however, in some cases be quite prominent. If pain be present, it is most acutely felt in the back, and an arching of the trunk backward may occur, called opisthotonos. Occasionally, emprosthotonos may take place, which is a bending forward, or to one side of the body, known as pleurosthotonos.

The muscles of the glottis may be affected, causing difficult noisy respirations and sometimes asphyxiation. In acute cases death occurs about the third day. In the less severe cases life may be prolonged for three or four weeks. Protracted cases afford the best hope for recovery.

The pulse is quickened during the seizures. The temperature varies,—it may be from 99° F. to 101° F., or after prolonged spasm hyperpyrexia may occur. High temperature may show itself just before the fatal issue. The mind is usually clear, although delirium has been noted in some cases. Profuse sweating is a prominent symptom of the disease.

The urine is scanty and extremely toxic, often containing albumin. When injected into animals, tetanus is produced.

Diagnosis.—In cases of trauma followed by tonic spasms the diagnosis is easy. Without the original focus, such as a wound, the diagnosis may sometimes be difficult.

Prognosis.—The prognosis is unfavorable. In the prolonged cases recovery may take place.

Treatment.—Great effort should be made to administer nutriment, and rectal alimentation should be resorted to. Chloral is of some use in that it produces sleep and occasionally relaxes spasm. Antitetanic serum has been of value in some cases. The wound should be thoroughly cleansed and treated antiseptically.

WEIL'S DISEASE.

Definition.—A rare infectious disease, characterized by fever, jaundice, gastro-intestinal disturbance, and great prostration, occurring in limited epidemics.

Synonym.—Acute infectious icterus.

Etiology.—Very little is known of the disease, it being extremely rare. It has occurred in limited epidemics in prisons and camps, and it appears that unsanitary surroundings may be a factor in its production. The disease occurs more often in men, usually in the summer months.

Pathology.—The lesions of granular degeneration occur-

ring in other infectious diseases are found present in Weil's disease. Degeneration of the kidney and liver is prominent. Bacteriologic investigation has yielded no positive results.

Symptoms.—The onset is marked by chill and a rapid rise in temperature. On the fourth or fifth day a remission usually occurs. The disease lasts three or four days longer than this, and defervescence comes on with a well-marked crisis. Occasionally, a relapse takes place. The pulse early in the disease is rapid and full, and as is so characteristic of jaundice, decreases in frequency as the disease advances. Prostration is a prominent symptom. Delirium and coma may occur, and there is usually mental dullness and apathy. Pains in the back of the neck and legs appear. The tongue is coated. There may be vomiting and either diarrhea or constipation, an enlarged and tender liver resulting in one-half of the cases.

The spleen is early enlarged. Jaundice is invariably present, and may be the first symptom of the disease. It varies in intensity in different cases. The urine is decreased in amount, containing bile pigment, albumin, and hyaline and granular casts. The stools are clay colored.

Complications and Sequels.—Catarrhal affections occur in the mucous membrane. Occasionally, purpuric manifestations are present. Secondary bubo appears in some cases.

Prognosis.—The prognosis is generally good, death, however, occasionally resulting.

Treatment.—The diet should consist exclusively of milk. Violent purging should be avoided. Alkaline mineral waters should be used. In the early stages fractional doses of calomel should be given. Enteroclysis of cold water is useful in many cases.

MALTA FEVER.

Definition.—A specific infectious disease, due to the micrococcus melitensis of Bruce, characterized by irregular fever, pain in the joints, free sweating, and other constitutional symptoms. The disease commonly lasts from three to six weeks. Relapses are frequent.

Synonyms.—Mediterranean fever; rock fever; Neapolitan fever; undulant fever.

Etiology.—The disease appears to be endemic in Malta and parts of the Mediterranean coast. It has recently been noted as occurring in some of the islands of the Gulf of Mexico and in Puerto Rico. It especially affects young adults.

The specific cause is the micrococcus melitensis of Bruce. (See p. 121.) Inoculation experiments have been successful in monkeys.

The **period of incubation** is from a few days to twenty or thirty.

Pathology.—The pathology is by no means settled. Perry, who made 100 autopsies of cases occurring in Gibraltar, found the typical lesions of enteric fever in every case. According to Bruce, the disease is rarely fatal, and upon autopsies no characteristic lesions are found.

Symptoms.—The onset of the disease is characteristic, marked by prodromes. Some authorities describe the onset identically with that of enteric fever. There are headache, general malaise, loss of appetite, epistaxis, sleeplessness, and thirst. Diarrhea is not common. After a day or two of these symptoms slight chills occur, followed by more or less fever. The fever is often of a remittent type, lasting from one to three weeks. A period of apyrexia then takes place, which varies in duration from one to three days, and is followed by a relapse, in which marked rigors, high fever, delirium, diarrhea (the stools sometimes containing blood), and great prostration occur. The relapse may last several weeks and then, after a remission of a week or two, a second relapse takes place with a return of the initial symptoms. In the second relapse there are marked sweating, muscular and joint pains are more pronounced, and the prostration is extreme. The disease now often terminates in recovery, or after a lapse of one or two months all the symptoms may reappear. In severe cases the temperature is high,—105° F.,—its course subcontinuous, and death may be due to hyperpyrexia. Complications are rare.

Diagnosis.—The disease may be differentiated from malaria by the microscopic examination of the blood.

Prognosis.—The mortality is low, estimated at about 2%. The course of the disease may be very protracted, and may last six months or longer.

Treatment.—The treatment is symptomatic.

PART II.

DISEASES OF THE CIRCULATION.

CONGENITAL MALFORMATION OF THE HEART.

Aside from abnormality in the size and position of the heart, defects in its development and structure occur. The heart may even be entirely absent in some individuals called acardiac monsters. There have been instances in which the heart has been found in the neck or in the abdomen. It may lie immediately beneath the skin, only covered by the pericardium. The heart has been found in the right side of the chest, with the blood-vessels reversed, the venous blood entering the left auricle, the pulmonary blood the right auricle, and the aorta having its origin at the right ventricle. This condition is known as dextrocardia. It is usual in such cases to have the abdominal organs also reversed, so that the liver is found upon the left side and the spleen upon the right side.

Complete absence or only a slight indication of the auriculoventricular septum has been observed, in which the entire heart may consist of practically only two chambers, with one blood-vessel springing from each. Defects in the interauricular and interventricular septa may be present. Stenosis of the pulmonary artery is the commonest of the malformations of the heart. Atresia or obliteration of the pulmonary artery is a much rarer condition.

The same conditions may be found in reference to the aorta, but are here even rarer than in the pulmonary artery. A patulous foramen ovale, or ductus Botalli, is by no means so rare. Valvular defects are common; thus, there may be but two semilunar cups, and sometimes four have been observed. A similar state of affairs has been met with in the mitral and tricuspid valves.

Congenital deformity and fetal endocarditis are the most prominent causes of these conditions. With dextrocardia the individual may be perfectly normal otherwise.

Symptoms.—The child is apt to be weakly, does not thrive well, and soon after birth presents signs of disturbance of the circulation, consisting of lividity of a bluish tint, affecting the face, hands, and feet. The respiration is labored and paroxysmal, and is apt to be increased by screaming, suckling, and exposure to a cooler atmosphere. The extremities are cold and the terminal phalanges of both hands and feet are often clubbed. The surface temperature is subnormal, although the rectal temperature may be 99° F.

Convulsions and cerebral attacks may take place independently of those induced by screaming, movement, and suckling, merging into coma, and often rapidly proving fatal.

Cyanosis is by far the most common symptom and occurs in about 90% of the cases. The blood shows marked alterations, the red corpuscles and the hemoglobin commonly being above normal.

Physical Signs.—A loud, prolonged systolic murmur is heard all over the cardiac region, being transmitted in all directions. If there be stenosis of the pulmonary artery, a thrill will be noticed in the pulmonary area.

Prognosis.—Grave in nearly all cases in which there is marked circulatory disturbance.

Treatment.—The child should be kept warm and exertion should be controlled. General good hygiene is necessary. The treatment by drugs is unsatisfactory.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Divisions.—Acute and chronic pericarditis.

ACUTE PERICARDITIS.

Definition.—An acute inflammation of the pericardium.

Etiology.—Etiologically, it may be divided into primary and secondary pericarditis.

Primary Pericarditis.—Primary pericarditis includes those cases which can not be referred to any previous underlying disease. It is occasionally found in persons without apparent

cause, but such cases are extremely rare, and in the present state of knowledge they had better be classed as cryptogenetic.

Those cases following injury, such as blows, wounds of the heart, or from internal causes, such as trauma of the esophagus, etc., may be classed as primary pericarditis.

Secondary Pericarditis.—By far the greater number of cases belong to the secondary variety. In these rheumatic fever plays the greatest causative rôle; it is associated with renal disease; with extension from neighboring structures, as from the pleura or diaphragm; with cardiac disease, such as myocarditis; occasionally, with direct disease of the aorta; with new growths, such as sarcoma; and, finally, it is frequently associated with various diseases, such as scurvy, tuberculosis, scarlet fever, diphtheria, septicemia, erysipelas, gout, or diabetes.

Pathology.—In acute pericarditis the visceral or the parietal layer may be involved; often the condition is general or may be localized to certain areas.

In the early stages the membrane becomes opaque, lusterless, and the surface roughened. Soon a fibrinous exudation appears upon the membrane or a serous exudate may result. The constant friction of the surface will cause a "honeycombed" condition of the fibrin or a "bread-and-butter" appearance.

If the irritant be severe, a hemorrhagic exudate is present or it may go on to suppuration and a pyopericardium be produced. The inflammatory process may subside at any time during the process of the disease and the exudation be absorbed or it may in some cases be retained. When absorption takes place, there are adhesions of the two surfaces, and fibrous connective tissue develops, called *adhesive pericarditis*. These adhesions may be general or local. General adhesive pericarditis, as a rule, causes hypertrophy of the heart; rarely, atrophy results from the contraction of the connective tissue.

Various micro-organisms are associated with acute pericarditis, such as the staphylococcus pyogenes aureus, albus, or citreus, the bacillus coli communis, the bacillus typhosus, Klebs-Löffler bacillus, the bacillus of Pfeiffer, and others.

Microscopically, the first change noticed in acute pericarditis is a parenchymatous degeneration of the endothelial cells. The blood-vessels under the endothelial layer show the

characteristic changes of acute inflammation. The exudation is formed as in all serous surfaces.

Symptoms.—It is impossible to diagnosticate acute pericarditis by symptoms, no matter how prominent they may be, without a careful physical examination. An accurate symptomatic description of this disease is, therefore, impossible. The disease commonly begins with severe pain, of a sharp-stabbing nature, in the region of the precordium. In some cases it may be referred to the epigastrium or left nipple. The pain is more or less continuous, but varies in severity in individual cases. It is increased by pressure or by manipulating the chest, as in percussion or palpation. In rare instances it may be absent altogether. The respiration becomes embarrassed. The pulse-rate rises as the disease increases in intensity; in the early stages it may be from 90 to 100, later it becomes more rapid, and in very severe cases may reach 160 a minute. In other instances the pulse-rate may be normal or the pulsus paradoxus may be observed, especially with effusion.

The respiration may be hurried and shallow. With effusion actual dyspnea is present, and the patient usually lies upon the left side. Cough, which is irritable and spasmodic, commonly appears. There may be difficulty in swallowing when the effusion is great, due to pressure on the esophagus. Fever of some degree is present in nearly all cases; it is not typical, varying from 100° F. to 103° F.

Nervous symptoms, such as headache, sleeplessness, restlessness, etc., occasionally appear. If the effusion be purulent, edema in the precordial region may be noted and a septic temperature may be present.

Physical Signs.—Inspection.—In the first or dry stage the apex-beat is seen in its normal position, and, unless there is an effusion, no bulging of the precordia will be noted.

Palpation.—Palpation may elicit friction fremitus. This may be felt at any part of the heart, usually, however, at the base. It does not necessarily occur with systole or diastole, but may be noted independently. It is more superficial than an endocardial thrill, and gives the palpating hand the sensation of being coarser.

Percussion.—Percussion in the first stage gives normal cardiac dullness. When effusion takes place, depending upon its size, a flat area is found with its base at the apex of the heart, merging into the normal cardiac dullness as the base of the heart is reached. If the effusion be enormous, the entire

precordial area may be flat upon percussion. The area is usually pear shaped, with the base downward toward the apex of the heart.

Auscultation.—In the first stage a friction sound is frequently heard, greatly varying in intensity. It may be described as rasping, coarse, or creaking in nature. It is limited to the precordial area, but may be heard at any part, most frequently, however, at the base. It usually occurs independently of systole or diastole; then again, it may be entirely systolic or diastolic. If the sound be ill defined, change in the position of the patient may sometimes make it prominent, such as sitting erect or leaning forward, thereby bringing the pericardium nearer the chest-wall, thus intensifying the friction sound.

It is increased by pressure with the stethoscope, which also increases the pain. As the effusion develops, the friction sound gradually subsides, disappearing last at the base of the heart. As absorption of the fluid takes place, the friction sound gradually reappears. In well-marked effusions the heart-sounds are distant, muffled, and may be entirely absent, particularly the first sound. The pulsus paradoxus frequently occurs, with large-sized pericardial effusions.

Diagnosis.—This depends upon the occurrence of the pain in the cardiac region with the friction sound and the knowledge of some infective process. A pleural friction sound occasionally gives rise to difficulty in diagnosis, but when the patient holds his breath, the pericardial friction sound continues, the pleural sound disappearing. Pleuropericardial friction sounds are sometimes encountered when both the pleura and pericardium are involved in the same process.

Differential Diagnosis.—Differential diagnosis must be made between a pericardial friction sound and an endocardial murmur. (See p. 90.)

Prognosis.—In uncomplicated cases recovery frequently takes place. Death may be due to a purulent effusion with symptoms of septicemia.

Treatment.—In the first stage ice-bags applied over the precordium are of use in relieving pain and quieting the cardiac action. Large blisters should not be used over the heart as they interfere with systematic examination. Small blisters at some distance from the precordium, however, may be of benefit, especially when there is an effusion. If pain be severe, opium in some form is necessary. In very large effusions

paracentesis is necessary, especially if the effusion be purulent; in this condition surgical interference is imperative. Gentle purges from time to time are useful. A mild nonstimulating diet is indicated.

SUPPURATIVE PERICARDITIS.

Synonym.—Pyopericardium.

Definition.—A purulent effusion in the pericardial sac.

Etiology.—Pyopericardium may be acute in its onset or, as is more commonly the case, may result from a subacute or chronic process. It is rarely the outcome of simple acute pericarditis, in which case a serous or a serous fibrinous exudate becomes purulent. In a large number of cases the pus may not be formed in the pericardium, but may be due to the accumulation of pus in a neighboring organ bursting into the sac, as in empyema of the pleura. It is always associated with pyogenic organisms. The longer a pericardial effusion remains unabsorbed, the more likely it is to become purulent. It may occur in connection with septicemia and pyemia, and occasionally in the course of the eruptive fevers, particularly smallpox; it may be associated with injuries and diseases of the bones.

If an abscess be formed in the myocardium, purulent pericarditis may result. In malignant endocarditis it is exceptionally found. The pericarditis associated with nephritis is supposed to have a special tendency to pus-formation. This has also been observed in tuberculous pericarditis. Pyopericardium is most common in young males.

Symptoms.—The symptoms are the same as those occurring in serofibrinous or fibrinous pericarditis.

Special symptoms are of no value in the diagnosis of pyopericardium, as it may occur with and without fever, and general septic phenomena; indeed, the septicemia may entirely overshadow the cardiac phenomena.

Prognosis.—The prognosis is very unfavorable.

Treatment.—The treatment is surgical.

CHRONIC PERICARDITIS.

Synonyms.—Chronic effusion; adhesive pericarditis.

Description.—Chronic pericardial effusion is extremely rare; it may be of a hemorrhagic or purulent nature, and is generally associated with new growths or tuberculosis.

Pathology.—As the adhesions of fibrous connective tissue develop, hypertrophy of the heart becomes marked. In some instances the "ox heart" is produced. Adhesions of the visceral and parietal layers are frequently general. The parietal layer may also become adherent to the chest-wall, diaphragm, or pleura.

Symptoms.—Pain may be a prominent symptom of this condition, and gives rise to attacks of angina pectoris. Inability to take a long full breath is sometimes a prominent feature, especially when the adhesions are extensive. There is marked disturbance of the cardiac action, and palpitation upon exertion is frequently a prominent symptom. Dyspnea is especially marked. Disordered function of the right ventricle is often prominent. It may come on gradually, becoming more and more pronounced, or may appear suddenly. This results in symptoms of dropsy, with congestion of the parts which relate to the portal circulation and the kidneys. The dropsy begins in the legs, but may involve the trunk and sometimes the upper extremities. Cyanosis and distended veins may be prominent symptoms, or there may be pallor with puffiness of the face. The liver is enlarged and can be palpated below the ribs, being painful and tender. Symptoms relative to the digestive tract may become prominent, and the urine is diminished in amount and often albuminous.

The symptoms just described may be entirely absent, even in well-marked cases.

Physical Signs.—The diagnosis of pericardial adhesions can be made only by the physical signs. These may be very prominent or very obscure. They are likely to be better marked if the adhesions are extensive. In some cases there is a distinct depression, more or less marked, in the precordial region, with narrowing of the intercostal spaces, the structures being drawn in by the external adhesions to the chest-wall. More commonly there is a bulging due to enlargement of the heart. The apex-beat presents differences in regard to its position and force: It may be noticed to the left of the parasternal line, even extending to the anterior axillary line, presenting all the evidences of great hypertrophy of the left ventricle. As a rule, it is carried outward, but it may be found in the fourth or fifth interspace, at the same time presenting evidences of great hypertrophy. On the other hand, the beat may be feeble or even imperceptible, with all the phenomena just described. This is attributed to the small

size and the weak action of the heart, being restrained by adhesions or great thickening of the pericardium.

Great variations of the situation, extent, and force of the impulse are noted. The impulse may extend upward through the entire precordial region, even reaching the second intercostal space. Occasionally, it is impossible to localize the apex-beat. It may be strong and superficial, the impulse being noticed close to the chest-wall, especially if the condition occurs in children. The rhythm is markedly distributed, giving rise to great irregularity of the heart's action. When the heart is enlarged, the impulse is increased, even passing beyond its normal limits, tending downward toward the right in consequence of enlargement (dilatation) of the right ventricle.

Systolic recession or retraction (dimpling) may take place. This sign is of great importance in pericardial adhesions. It may occur at the apex and be associated with the apex-beat, or may appear in one or more intercostal spaces to the left of the sternum, especially in the third, fourth, or fifth interspace. The movement may be wavy.

When there is retraction at the posterior or lateral portion of the thoracic walls (interspaces), adhesions have usually formed with the diaphragm. They are noted during systole. This is known as "*Broadbent's sign.*" Broadbent described it as follows: "In cases of adherent pericardium, marked systolic retraction of some of the lower ribs on the lateral or posterior aspect of the thorax may sometimes be seen. This phenomenon is best seen when the patient is sitting up in a good light, and the movements of the chest are carefully observed from a short distance off, first from the front and then from the lateral aspect. When a pulsatile movement is seen over the lowest part of the left side posteriorly, it may at first sight appear to be expansile. On a more careful scrutiny it will be found that there is a tug on the false ribs during the cardiac systole and a sharp rebound during diastole, which can be felt as well as seen when the hand is laid flat upon the chest-wall at the spot; it is more marked when deep inspiration is made; it may be seen occasionally, not only upon the left side, but also on the right, especially if the patient leans over to the left."

Sometimes there is diastolic shock. In well-marked cases it may be felt as a distinctive jerk or blow, occurring over the

precordial area. It is a favorable sign in adherent pericardium, showing compensatory hypertrophy.

Auscultation.—The signs upon auscultation may consist in the pericardial friction sound, which may remain. The first sound is often abnormal in character. It is sharp and frequently valvular, or may be dull and muffled; occasionally it is prolonged and reduplicated. The second sound may also be reduplicated, and may cause diastolic shock. If adhesions form with the pleura, the pericardial friction sound may occur with inspiration and expiration.

The pulsus paradoxus has been noted in this condition; during diastole there is sometimes sudden collapse of the veins of the neck; this is known as "*Friedreich's sign.*"

The pulse will give the character of the condition of the ventricle: If the ventricle be markedly hypertrophied, the pulse will be full and strong; on the other hand, if there be great dilatation of the ventricle, or if the contraction interfere with the movement of the heart, it is feeble.

Diagnosis.—A positive diagnosis of adherent pericardium can only be made in the presence of the physical signs just enumerated. Severe heart symptoms in young persons, without valvular murmurs, point to pericardial adhesions; whereas in persons of more mature age it is a sign of cardiac degeneration.

Prognosis.—Pericardial adhesions are not amenable to treatment. If slight, they may be of very little consequence. When they are marked and severe, the disease is one of great seriousness. Valvular disease complicating this condition adds to the gravity of the case.

Treatment.—Rest in the recumbent position is most important in preventing the great hypertrophy that is apt to take place. Cardiac tonics may be useful in some cases, but digitalis should be avoided unless signs of failure of compensation occur. Symptoms must be dealt with as they arise.

HYDROPERICARDIUM.

Synonym.—Dropsy of the pericardium.

Pathology.—The amount of fluid contained in the pericardium may vary from a slight increase over the normal to a liter or more. It is a transudate; the fluid is clear, straw colored, and of low specific gravity,—usually below 1018,—and the amount of albumin present is slight. It frequently

occurs in connection with general dropsy, as from kidney disease or valvular heart disease. It may also occur with anemia, scurvy, tuberculosis, or allied conditions.

In all the above-enumerated conditions, especially kidney disease (being most frequent in the chronic form of parenchymatous nephritis), fluid is also found in the other serous cavities.

Interference with the local circulation of the pericardium, such as thrombosis in the pericardial veins or from pressure, rarely gives rise to hydropericardium.

Symptoms.—This condition can only be differentiated from pericardial effusion by the absence of friction sounds and the history of the case; the other signs and symptoms being similar to pericarditis with effusion.

Treatment.—The treatment consists in attention being directed to the cause.

HEMOPERICARDIUM.

Synonym.—Blood in the pericardium.

Pathology.—This condition is rare, and results from external injury; frequently, however, it is caused by an aneurysm of the aorta rupturing into the pericardial sac; rarely aneurysm of the heart itself or from the coronary arteries may occasion it. Hemorrhages may result from purpura and allied conditions. The hemorrhage may occur from a small leak, the blood coagulating; or it may be copious, distending the pericardial cavity suddenly.

Symptoms.—Death may ensue rapidly if the quantity of blood poured into the pericardium be large. If it takes place more slowly, there may be pain, faintness, syncope, dyspnea, with rapid, feeble pulse due to lowering of the blood pressure, and the physical signs of accumulation of fluid into the pericardial sac. From lowering of blood pressure a decrease in the amount of urine, or anuria, may result.

Prognosis.—The prognosis is hopeless.

Treatment.—The treatment is entirely symptomatic.

PNEUMOPERICARDIUM.

Synonym.—Gas in the pericardium.

Description.—This condition is extremely rare and results from perforation through the esophagus, lungs, or stomach,

or from an external wound. Ulceration from a tuberculous cavity establishing communication with the pericardium may be a cause. In rare instances the gas-producing bacillus (bacillus aerogenes capsulatus) will be found associated. If it results from ulceration, pus may be found in the pericardial sac. Great distention of the pericardium will cause pressure upon surrounding structures.

Symptoms.—If gas collects rapidly, there will be great precordial distress, dypsnea, cyanosis, and collapse, with irregular and feeble pulse. High fever with chills and profuse sweating may accompany this condition.

Physical Signs.—The physical signs will depend upon the amount of gas in the pericardial sac. There may be bulging or fullness in this region. The apex-beat may be weak or absent and better felt as the patient bends forward. A succussion splash may occur upon shaking the patient if fluid be also present in the pericardium.

Percussion may develop a note of metallic quality. The signs upon auscultation vary greatly. Metallic tinkling may be noticed; bell tympany has been observed in some cases.

Prognosis.—The prognosis is grave.

Treatment.—The treatment consists in the administration of stimulants and sedatives. The question of operation presents itself, and in suitable cases the gas may be let out by a fine trocar with the patient in the recumbent posture. This condition must be dealt with from a surgical point of view.

NEW GROWTHS AND PARASITES OF THE PERICARDIUM.

Tuberculosis of the pericardium has been described under Tuberculosis. (See p. 278.)

Carcinoma is always secondary, and occurs from extension of neighboring organs. Sarcoma is met with, especially of the melanotic variety, and is most frequently secondary. Hydâtids are extremely rare.

ACUTE ENDOCARDITIS.

Definition.—Inflammation of the internal lining membrane, or endocardium of the heart. This condition especially affects the valve segments. The walls are occasionally affected, when the condition is known as *mural endocarditis.*

Two varieties are recognized, *acute* and *chronic*. The acute is divided into two forms,—simple acute or benign, and the malignant or infective.

SIMPLE ACUTE ENDOCARDITIS.

Synonyms. — Benign, papillary, verrucose, or rheumatic endocarditis.

Etiology.—The greatest number of cases are the result of acute rheumatic fever. This has been variously estimated as between 20% and 30% of all cases of rheumatic fever. It occurs more frequently in children than in adults, and usually appears during the first attack of acute rheumatic fever. If it does not result from the first attack it is almost invariably the consequence of the second or third attack. It appears in the milder as well as in the severer cases, *and very frequently in patients who have not manifested prominent joint affection.* Chorea sometimes gives rise to acute endocarditis. It appears in the course of the acute exanthematous fevers, scarlet fever occupying the first place. It is occasionally due to diphtheria, enteric fever, measles, smallpox, pneumonia, erysipelas, puerperal and septic diseases, and sometimes as the result of gonorrhea. Acute and chronic tuberculosis, gout, Bright's disease, and diabetes are etiologic factors. Trauma is also a cause of acute endocarditis. It must be stated that occasionally endocarditis arises apparently cryptogenetically. It occurs in fetal life, but here it usually attacks the right side of the heart. Age is important, as the affection shows itself most frequently between the ages of fifteen and forty, but is rare in old people, in whom valvular lesions are most often due to atheromatous changes.

Finally, endocarditis may be secondary from extension of the disease from the myocardium or aorta.

Pathology.—The lesions are usually situated in the left side of the heart. The endocardium lining the valves is affected in the following order of frequency: First, the mitral; second, the aortic, and on the right side of the heart, the tricuspid; and, lastly, the valves of the pulmonary artery. The endocardium of the valves is more frequently affected than the mural lining.

The lesions are small vegetations, varying in size from one to four millimeters in diameter. They are small, warty, or cauliflower excrescences,—hence the name "*verrucose*,"—

and are most frequently situated on the valves at the line of contact, which in the mitral and tricuspid valves is the auricular surface and in the aorta and pulmonary valves the ventricular. Usually, micro-organisms are associated with simple endocarditis. The following are the most important: Staphylococcus, streptococcus, gonococcus, diplococcus of pneumonia, Eberth's bacillus, bacillus coli communis, and the bacillus of Pfeiffer.

Microscopically, the first change noted in the endocardium is the granular degeneration of the endothelial cells. In the subendothelial tissues, blood-vessel changes that accompany inflammation are noted. Coagulation necrosis and the proliferation of the fixed cells are found. The inflammatory exudate in the subendothelial tissues and the fibrin upon the free surface causes the bulging or warty excrescence. Upon this projection fibrin from the blood stream is also deposited. Intermingled with the exudate the various micro-organisms just enumerated may be present.

Simple acute endocarditis may terminate in organization, but it is questionable whether complete recovery without deformity of the valve ever results. Most frequently, the condition terminates in chronic sclerotic endocarditis with marked deformity of the valve segments, which remains permanently. Occasionally, the simple acute variety terminates or merges into the malignant. Destruction of the warty excrescence, such as breaking off and forming an embolus, rarely happens in the simple form.

Symptoms.—The disease may come on without giving rise to any symptoms, the subjective phenomena varying greatly in intensity and the physical signs usually only appearing after the disease has become chronic. Attention may only be attracted to the endocardium by an attack of hemiplegia due to embolism. If the disease follow acute rheumatic fever, the symptoms will be largely those due to the infection, and the endocarditis can only be diagnosticated by the physical signs.

In other instances during the attack of an infectious disease symptoms of oppression, with uneasiness and pain in the cardiac region, may appear. The pulse may become small and quick, and the heart's action tumultuous. Dyspnea upon exertion is a symptom of some importance. If pericarditis complicate this condition, pain is likely to be a prominent symptom.

Physical Signs.—Physical signs may be very marked in some cases, and in others absent altogether.

Early on inspection no change is usually noticed, and the trustworthy and important physical signs can only be discovered upon auscultation. As the mitral valve is most frequently involved, a systolic murmur, which has its maximum intensity at or near the apex and is transmitted to the left axilla and often to the angle of the scapula, shows that mitral regurgitation has taken place. The murmur is at first soft and blowing in character. Its occurrence may be preceded for a few days by an impurity and a prolongation of the first sound. In children the signs are more marked, as a rule; tumultuous, quickened, and uneven heart-sounds, which are sometimes reduplicated, appear.

If the endocarditis affect the aortic valves or if the vegetations be small, no special signs may be present; or, again, a diastolic murmur will appear at the aortic cartilage, which is transmitted down the sternum, showing that aortic incompetence has taken place. Stenosis of either the mitral or aortic valve is rare. This condition most often occurs in the chronic form of endocarditis.

In rare instances the right side of the heart may be affected and murmurs, which relate to the tricuspid or pulmonary valve, make their appearance.

Complications.—The most frequent complications are pericarditis, myocarditis, and occasionally pleurisy and pneumonia. Emboli and infarcts are more likely to arise in malignant endocarditis, in which case the valve may entirely recover its normal function. In the majority of instances, however, the physical signs, when once well marked, do not disappear.

If compensation be complete, symptoms do not arise, and it often happens that only with failing compensation attention is directed to the heart.

Death may result from large pericardial effusions, hyperpyrexia, or toxemia, or the disease may be converted into malignant endocarditis.

Diagnosis.—The diagnosis depends entirely upon the physical examination. The presence of well-developed murmurs, which remain stationary and which are conducted in definite directions, all indicate some permanent valve defect. In the absence of a well-defined murmur in the course of acute rheumatic fever, any symptoms that point directly to the heart should be carefully investigated, for even in the mildest cases

of acute rheumatic fever, in which little or no joint affection occurs, the heart is extremely likely to suffer.

Differential Diagnosis.—The differential diagnosis between an organic murmur and a functional murmur may occasionally give rise to difficulty. The functional murmur is most frequently heard at the left base; it is not transmitted; it is always systolic in time, and accompanies anemic conditions.

The commonest seat of acute endocardial disease is at the mitral valve, the murmur being heard at the apex and transmitted to the left axilla, and often to the angle of the scapula.

Prognosis.—Death in simple endocarditis during the acute stage is due either to the severity of the primary disease or to some complication. A large number of patients recover from the acute attack, but become subjects of chronic valvular disease. When acute endocarditis occurs in persons affected with valvular disease, the prognosis is more serious, as in fresh outbreaks the disease is likely to be infective or malignant.

Treatment.—The treatment consists in absolute rest in the recumbent posture. Local applications of ice, especially when pericarditis is present, are of use. Digitalis should only be given for definite and fixed indications, and is not required unless the pulse becomes quick and small or irregular, or signs of failing compensation occur. Strychnia is of benefit in this condition. If anemia persists, iron with quinin and arsenic will be found valuable.

INFECTIVE ENDOCARDITIS.

Synonyms.—Malignant endocarditis; ulcerative endocarditis; mycotic endocarditis.

Etiology.—The conditions that give rise to simple endocarditis also give rise to malignant endocarditis. This variety of endocarditis is due either to the great amount of toxemia or the great preponderance of organisms in the process.

Pathology.—The pathologic changes that occur in infective endocarditis are very similar to those of simple or benign endocarditis. The virulence or the number of micro-organisms is probably greater, the process more rapid, and destruction of tissue more pronounced, so that it is common in this condition to have infected emboli swept through the blood-current, lodging in the various organs, and producing infarcts and embolic abscesses. The endocardium on the left side of the heart

is, again, most commonly involved; that lining the valves more often than that of the wall, and the same order of frequency occurs regarding the valve affected as in the simple acute variety.

The vegetations are frequently large, projecting into the cavities of the heart, with a ragged, irregular surface, and very friable. Very often ulcers are found as a result of the breaking down of the vegetation, the myocardium being involved in not a few cases. Abscesses of the myocardium may form, as a consequence of which aneurysm or rupture of the heart may result. The chordæ tendineæ and papillary muscles are often destroyed.

Microscopically, the same changes will be noted as in acute simple endocarditis, except that the process is more extensive. Through extension the pericardium may be inflamed. Various micro-organisms and their toxins, especially the latter, are often present in the blood. Leukocytosis is almost constantly found.

Symptoms.—The symptoms vary, and the cardiac phenomena may be slight, insignificant, or even absent. In other cases the symptoms are pronounced and readily call attention to the fact that the heart is affected.

On account of the great variety of symptoms four special types have been recognized. The symptoms of each type, briefly considered, are as follows:

Septic or Pyemic Type.—This occurs during the course of septicemia, pyemia, sometimes in the puerperal state, or in the course of any severe septic infection.

The onset is acute, without prodromes, the disease being ushered in by chills, fever, and sweating, which may be repeated after a longer or shorter interval. The temperature, as a rule, is of the remittent type. The skin may show patches of erythema or purpuric manifestations, or may contain superficial collections of pus. The pulse is rapid and feeble; the respiration, shallow and quickened. Headache, delirium, and coma are the nervous symptoms most often present, and at times hemiplegia due to cerebral embolism occurs. The tongue is dry, brown, and furred. Anorexia, with vomiting and great tympanites, is present.

Abscesses are likely to form in various organs and tissues. The patient may present no cardiac signs; or, on the other hand, there may be murmurs, which vary in intensity and duration. Albuminuria, pain and swelling of the joints, with

suppuration, sometimes take place. Death occurs in from one to two weeks.

Typhoid Type.—This closely resembles enteric fever, showing the same conditions of the tongue, the same abdominal symptoms, and marked nervous symptoms. Not infrequently rigors are present; petechiæ occur, and optic neuritis is a symptom. This rarely occurs in enteric fever.

The physical signs relating to the heart may be very indefinite or may be absent. The temperature is irregular, and rigors occur throughout the attack, followed by profuse sweating. Embolism of the brain, kidney, or spleen is common. The disease may last from two to four weeks, terminating fatally.

Cerebral Type.—The affection shows itself by symptoms relating to the cerebrospinal system, particularly headache, somnolence, rapidly passing into unconsciousness and coma, with delirium and convulsions. Chills and fever are rarely present, but embolic processes may call attention to the heart, although the cardiac phenomena be not well marked.

Cardiac or Malarial Type.—The majority of cases of ulcerative endocarditis are of this variety and appear in persons affected by some form of chronic endocarditis. It may run a subacute or chronic course, and last for months and even a year.

Recovery is extremely rare, most cases proving fatal. The onset is insidious with prodromes or general malaise. A slight increase in the temperature may be one of the earliest symptoms. The disease may resemble rheumatic fever with pain in the joints, but in either case chills soon occur, with fever and sweating.

The temperature may show either a remittent or intermittent type, and 104° F. or even higher ranges have been noted. The chills occur at irregular intervals, and several days or even weeks may elapse before a second rigor appears. In this variety embolic processes are most likely to occur. These may take place in the peripheral arteries, in the middle cerebral artery, or any of its branches, sometimes causing permanent hemiplegia. Splenic infarct is common, occasionally without giving rise to symptoms. A symptom that should at once call attention to this condition (splenic infarct) is a sudden sharp pain in the region of the spleen with an increase in splenic dullness upon percussion. Renal and pulmonary infarcts also occur. Pains in the joints are common; petechiæ and hemorrhage from the mucous membranes are met with,

especially if the aortic valves are affected. Hemorrhages into the retina and optic neuritis are extremely common in this condition. The urine shows traces of albumin and blood with epithelial and granular casts. The liver and spleen remain enlarged. The bowels may be constipated, but profuse diarrhea with hemorrhages from the bowel occasionally occur.

Symptoms relating to the heart are most common in this condition, and murmurs at any of the valves may appear. These murmurs are characterized by their changeable nature, now appearing at the mitral and now at the aortic valves. They may be either soft or harsh in character. Palpitation, cardiac pain, and excessive dyspnea may occur.

Complications.—Pneumonia, pleurisy, pericarditis, cerebral hemorrhage and aneurysm are the most frequent complications.

Diagnosis.—The diagnosis depends upon the presence of fever, which may be either of a remittent or intermittent type, with chills and marked sweating; anemia is constantly present. Enlargement of the spleen and liver occurs. Changes take place in the retina with optic neuritis, as do also hemorrhages into the mucous membranes and skin. Hematuria from renal infarcts and other signs of infarcts, with bacteriologic examination of the blood with the inconstant and changeable valvular murmurs, and leukocytosis are characteristics of this condition. The appearance of a right-sided involvement should call attention to infective endocarditis, as this is extremely rare in the simple variety.

Prognosis.—The prognosis is very unfavorable.

Treatment.—No drugs are known that have any influence on this condition. If the infection be due to the streptococcus, the antistreptococcic serum will usually be of value, several successful cases having been reported. The treatment should be supportive and tonic. The measures that have been advocated in simple endocarditis may be of use also in this condition.

CHRONIC ENDOCARDITIS. SCLEROTIC ENDOCARDITIS. MITRAL INSUFFICIENCY.

Synonyms.—Mitral incompetency; mitral regurgitation.

Definition.—A diseased condition of the mitral valve or of the left auriculoventricular opening, forcing the blood into the left auricle with the contraction of the ventricle.

Etiology.—Acute rheumatic fever is the primary cause leading to the greatest number of cases of mitral insufficiency.

Extreme anemia with changes in the muscular substance of the walls of the left ventricle, diminishing its contractile power, may result in this condition. It may be due to stretching of the auriculoventricular orifice accompanying dilatation of the left ventricle, which is secondary to changes at the aortic orifice. Occasionally, disease of the columnæ carneæ and chordæ tendinæ, which allow the flaps of the valve to pass beyond the plane of the orifice, gives rise to mitral incompetency. It is encountered at all ages, but is most frequent in the young. It is most often the result of acute endocarditis passing into chronic endocarditis.

Pathology.—The most frequent lesion is a shortening or thickening of the chordæ tendinæ, preventing proper closure. Rarely, there may be stretching of the chordæ tendinæ, causing a lengthening and an overlapping of the mitral segments. Other sclerotic changes, such as one or more segments becoming united, may reduce the elasticity, often converting them into a firm band.

Calcareous infiltration, especially at the base of the mitral segments, is quite common. Acute or chronic myocarditis may prevent proper closure. As a result of the improper closure blood regurgitates into the left auricle during the ventricular systole, first producing increased tension in the pulmonary veins with dilatation of the left auricle, which is soon followed by hypertrophy of this chamber of the heart. The left ventricle now receives more blood and hypertrophies. The increased pressure in the pulmonary circulation throws more work upon the right ventricle, which, in turn, hypertrophies. In this way compensation may be maintained. Sclerotic changes in the pulmonary artery and veins and brown induration of the lung result from the increased tension in the pulmonary circulation.

Symptoms.—Unless rupture of a valve or of the chordæ tendinæ occur suddenly, the symptoms may be latent for a long time. Especially is this the case if compensation be well established. Only with failing compensation do the following symptoms make their appearance: dyspnea, especially upon exertion; cough; expectoration which is frequently tinged with blood; decreased amounts of urine containing albumin; and gastro-intestinal disturbances. The pulse may remain regular in force and rhythm. This condition must be diagnosticated by the physical signs.

Physical Signs.—*Inspection.*—The area of cardiac impulse

is greatly increased and more or less distinct, depending upon the hypertrophy of the right ventricle. Especially in children will there be bulging of the precordium, with a heaving of the thoracic wall.

Palpation.—The apex-beat is displaced to the left; the impulse is diffused and more or less forcible, depending upon whether the hypertrophy of the right or left side predominates. A systolic thrill, most distinctly felt at the apex, in rare instances may be found. This is almost always associated with a presystolic thrill, showing coexisting mitral stenosis.

Percussion.—The area of cardiac dullness is increased laterally and downward.

Auscultation.—A systolic murmur, which has its maximum point of intensity at or near the apex, being transmitted to the left axilla and angle of the scapula, is diagnostic of this condition. It is usually soft and blowing in character, and may become distinctly musical. Accentuation of the second pulmonary sound is characteristic. This is heard particularly at the second left interspace. Mitral stenosis frequently coexists with this condition. In such cases a presystolic murmur and a presystolic thrill will be present at or near the apex.

Diagnosis.—The diagnosis depends upon the recognition of a systolic murmur at or near the apex, transmitted to the left axilla, often to the angle of the scapula, with accentuation of the second pulmonary sound, and changes in the heart-muscle.

MITRAL STENOSIS.

Synonyms.—Narrowing or obstruction of the mitral valve.

Definition.—In this condition the blood passes with difficulty from the left auricle to the left ventricle. Frequently stenosis coexists with insufficiency of the mitral valve, and it is questionable whether stenosis occurs separately and apart from mitral regurgitation.

Etiology.—Mitral stenosis is almost invariably the result of valvular endocarditis occurring in children and young adults. It is rarely seen after the fiftieth year. It is nearly twice as prevalent in females as in males. A limited number of cases seem to be congenital. Acute rheumatic endocarditis is the most frequent cause.

Pathology.—Narrowing of the mitral orifice results from sclerotic changes in the valve segments so that they are united; contraction results, and there is produced a condition

commonly called a "buttonhole" mitral valve. This is almost invariably associated with some degree of incompetence, as the valve segments are entirely obliterated and a band-like ring is present. As a result of this narrowing, dilatation of the left auricle, which is soon followed by hypertrophy, occurs. This hypertrophy is marked, the muscular wall being three or four times its normal thickness.

The increased pulmonary tension causes more work for the right ventricle, and hypertrophy of this chamber follows, which is marked in this lesion. The left ventricle, in a majority of the cases, is not hypertrophied, because it has less work thrown upon it, receiving even less blood than normal. Atrophy of this chamber has been noted. Similar changes result in the pulmonary arteries, veins, and lung as have been described in mitral incompetency.

Symptoms.—Few subjective symptoms are present unless compensation is ruptured. The patient is usually anemic, and precordial pain may be a symptom. The pulse is small, feeble, rapid, and irregular. Attacks of fainting may take place, due to the brain not receiving sufficient blood.

Physical Diagnosis.—Inspection.—The cardiac impulse is feeble and indistinct. It is rarely seen to the left of its normal area, and is usually most distinct in the fourth interspace near the sternum.

Palpation.—The apex-beat is feeble. A distinct, purring, presystolic thrill, at or slightly above the apex, is frequently communicated to the hand. It may begin during diastole and run up to the ventricular systole, when it ceases abruptly.

Percussion.—The area of the cardiac dullness may be slightly increased to the right and upward.

Auscultation.—A loud, blubbering, presystolic murmur at or near the apex is diagnostic of this condition. The murmur is longer in duration than any other murmur. It is synchronous with the purring, presystolic thrill, and is heard slightly above the apex, being louder when the patient is sitting than when reclining in a recumbent position. *It is not transmitted.* The second pulmonic sound is accentuated and occasionally the second sound is reduplicated. When mitral incompetency coexists, a systolic murmur is also present, the presystolic and systolic murmurs constituting one. The presystolic murmur is always harsher and longer than the systolic murmur.

Diagnosis.—The diagnosis depends upon the feeble, small, rapid pulse, the presystolic thrill and presystolic murmur,

having its point of maximum intensity slightly above the apex, with a marked accentuation of the second sound. The presystolic murmur is not transmitted.

AORTIC INSUFFICIENCY.

Synonym.—Aortic regurgitation or incompetency.

Definition.—This condition is produced by an abnormal state of the aortic valves or dilatation of the aortic orifice, which prevents complete closure during the cardiac diastole, and allows a backward current of blood to flow from the aorta into the left ventricle.

Etiology.—It is most frequently found during the middle periods of life in vigorous athletic persons. Acute endocarditis is rarely a cause, unless it be due to the malignant form, in which case death rapidly results. The common causes are fibroid change leading to thickening, induration, and contraction of the valves. Syphilis is often the cause of this condition. Congenital insufficiency is very rare.

This lesion frequently occurs in persons subjected to violent muscular exertion—those engaged in the various trades and occupations that require heavy lifting and great muscular force. Athletes are often subject to this form of valvular disturbance, in whom it may lead to sudden death.

Pathology.—Aortic insufficiency may result from congenital defects, as absence of one of the cusps. Sclerotic changes often develop, causing a shrinkage of one or more of the segments or their complete obliteration into a fibrous mass, producing stenosis as well as insufficiency. Calcareous infiltration is very commonly associated with this sclerotic change. Atheroma of the aorta may interfere with the closure of the valve. Aneurysm of the first part of the arch of the aorta causes a relative insufficiency. Rupture of a leaflet may produce it. As a result of insufficiency, the left ventricle is called upon to do more work, as the arterial blood flows into the ventricle during diastole, so that it must handle this as well as blood received from the auricle. Hypertrophy, in consequence, follows; which often assumes enormous proportions.

The left auricle has more work thrown upon it, and as a consequence hypertrophies; finally, the right ventricle shows hypertrophy as a result of the increased pulmonary tension. The hypertrophy of the heart in aortic regurgitation reaches immense proportions, the heart frequently weighing 600

grams. This is called the ox heart (" cor bovinum "). Dilatation of the aorta and carotids as well as aneurysm may result. Sclerotic changes are sometimes found in the mitral valve, and the papillary muscles may be flattened. Sclerosis of the coronary arteries, followed by changes in the myocardium, such as fatty degeneration, frequently take place. Chronic interstitial myocarditis may also be found, as well as general sclerotic changes in the arterial system and kidneys.

Symptoms.—This condition may exist for years without giving rise to symptoms, and only with loss of compensation do they appear. Palpitation, especially upon exertion, is usually the earliest indication. The pulse in this disease is characteristic. It is known as the " Corrigan," " receding," or " water-hammer " pulse. It is large and distinct and suddenly projects against the finger; at the time when arterial tension sinks, the impulse vanishes abruptly. Its characters become especially noted when the arm is raised above the head, as this favors the receding character. It is always regular in rhythm while compensation lasts. A delay in the pulse is noted. This pulse is due to the nonsupport of the great quantity of blood thrown out by the hypertrophied left ventricle, the aortic valves not closing perfectly and permitting regurgitation. Angina pectoris sometimes occurs in this condition. Tinnitus aurium may also be a symptom.

Physical Signs.—Inspection.—Inspection shows a forcible and increased cardiac impulse. The apex-beat is displaced downward and to the left, often reaching the seventh interspace in the anterior axillary line. The vessels of the neck are seen to pulsate forcibly, as do also the superficial vessels, particularly the temporals. The capillary pulse occurs in aortic incompetency.

Palpation.—This confirms inspection. The impulse is forcible, heaving, and may be perceived over the entire cardiac area.

Percussion.—The area of cardiac percussion is increased downward and to the left.

Auscultation.—A diastolic murmur, with or taking the place of the second sound, heard with greatest intensity at the aortic cartilage, and sometimes at the ensiform, transmitted down the sternum, is characteristic of this condition. It is sometimes heard loudest at the xiphoid cartilage. Occasionally it is transmitted toward the apex. This murmur is more greatly diffused than any other organic murmur, and may

occasionally be heard even in the arteries of the neck. It may be soft and blowing, or rough, and occasionally musical in character. A presystolic murmur, known as the "Flint murmur," is sometimes heard at the apex. This is due to one of two causes, or a combination of both. According to Sansom and Potain, "It may be due to the impingement of the refluent aortic current on the anterior mitral curtain before it is made taut, whereby vibrations are set up in the valve itself, or by bulging the valve the orifice is practically narrowed" (Allbutt). The first sound is loud and booming in character, as occurs in all cases in which there is hypertrophy of the ventricle. In this form of valvular disease sudden death is likely to take place from rupture.

Diagnosis.—This depends upon the characteristic pulse (Corrigan pulse), the diastolic murmur heard at the second right costal cartilage, transmitted down the sternum, and the great hypertrophy of the left ventricle, with the capillary pulse.

AORTIC STENOSIS.

Synonyms.—Aortic narrowing; aortic constriction.

Definition.—A diseased condition of the aortic valve, causing more or less narrowing of this orifice with hypertrophy of the left ventricle. In nearly every instance this valve lesion is associated with some degree of insufficiency.

Etiology.—It is a disease of advanced life, and is often associated with arteriocapillary fibrosis. Rheumatic fever may give rise to the double lesion at the aortic valve. Increased tension of the aorta and prolonged muscular exertion may be the causative factor in the changes at the aortic orifice, especially in persons of a gouty diathesis; particularly is this so in females. Aortic valvular disease is not commonly of rheumatic origin.

Pathology.—Fibrous changes along the edges of the aortic cusps, extending throughout the valve segments, may cause a slight degree of obstruction, as well as incompetency. The most marked obstructive lesion is produced by fibrous induration at the base of the valve cusps, causing a contraction and thereby narrowing the orifice. Calcareous infiltration into the sclerotic area not infrequently follows. Recurrent acute endocarditis may follow upon an old lesion.

As a result of the lesion of stenosis, the left ventricle first dilates and then is followed by marked hypertrophy, which,

next to the lesion of aortic regurgitation, shows the greatest thickening of this part of the heart. Subvalvular stenosis, or a lesion of the endocardium, in the left ventricle, causing some obstruction to the outflowing blood, has been noted. Aortic stenosis may result from fetal endocarditis.

Symptoms.—Few if any symptoms occur, provided compensation be established, and quite extensive stenosis is not incompatible with a fair degree of health, if there be sufficient hypertrophy of the left ventricle, enabling the heart to fill the arterial system and relieve pulmonary pressure.

Pallor, cold hands and feet, shortness of breath upon exertion, impaired nutrition, vertigo, and nausea may be symptoms. The pulse is normal or below the normal in frequency, but may be quite strong and forcible (pulsus tardus). It may be quite regular in rhythm, but occasionally it is intermittent and jerky. Embolic processes in the brain are more often associated with aortic stenosis than with any other chronic valvular lesion.

Physical Signs.—Inspection.—The area of cardiac impulse is increased, the apex being displaced outward and downward. In old persons in whom emphysema is prominent there may be no visible impulse.

Palpation.—Palpation confirms inspection. A systolic thrill is often felt in the region of the second right intercostal space, or in the neck. This thrill is usually well marked.

Percussion.—The area of cardiac dullness is increased to the left and downward.

Auscultation.—A systolic murmur is heard at the second right costal cartilage and transmitted into the vessels of the neck. The first sound of the heart may be heard, but the murmur either replaces or obscures it. It is loud and harsh in character, and usually loudest at the beginning of the systole. The second sound of the heart is often obscured. This murmur follows the direction of the blood current, and may be heard over the thoracic and abdominal aorta or other large arteries.

Diagnosis.—The diagnosis depends upon the systolic murmur heard at the second right costal cartilage, transmitted into the vessels of the neck, accompanied by a systolic thrill in the aortic area, with a hard, commonly slow pulse, and considerable hypertrophy of the left ventricle.

It should be remembered in this connection that anemic murmurs, which are also systolic in time, may be heard in this area. Murmurs which are due to dilatation and roughening of

the aorta also occur in this area, but in none of these conditions is a systolic thrill present.

TRICUSPID INSUFFICIENCY.

Synonyms.—Tricuspid incompetency; tricuspid regurgitation.

Etiology.—This may be the result of an endocarditis which has thickened and puckered the valve, often congenital. Any condition of the lungs which produces dilatation and hypertrophy of the right ventricle may lead to it. It is met with in pseudohypertrophic pulmonary emphysema and in advanced cases of chronic bronchitis. It is most frequently secondary to some mitral lesion.

It occasionally is the result of an infective endocarditis, occurring during the course of gonorrhea, puerperal fever, etc. In such conditions it is primary.

Pathology.—The valves may be shrunken and thickened, due to chronic changes produced by rheumatic fever, but more often the consequence of atheroma following ventricular pressure. This may result in mitral disease, disease of the lung, or from chronic renal disease (granular kidney).

Obstruction in the pulmonary circuit causes dilatation of the right ventricle, and in this way produces relative incompetency of the tricuspid valve. General venous obstruction may follow, and symptoms of cyanosis and dropsy make their appearance.

Symptoms.—The symptoms may be entirely masked by an accompanying affection of the left side of the heart. It will be noticed, however, that as soon as the valve becomes insufficient the venous return is impeded. Cardiac palpitation, dyspnea, and irregularity in force and rhythm of the heart occur. The area of splenic and hepatic dullness is increased.

Dyspeptic symptoms become marked, and obstinate constipation is the rule. Headache, dizziness, and vertigo are symptoms showing cerebral congestion. While the patient is in the recumbent posture the face becomes turgid and cyanotic. The urine is scanty, dark, and of high specific gravity, often containing albumin. Dropsy shows itself in the later stages of the disease, beginning in the feet and continuing until there is anasarca.

Physical Signs.—Inspection.—The area of the cardiac impulse is very extensive. It may be seen from the left nipple to the ensiform cartilage. Pulsation may be seen in the jugu-

lar veins. The veins of the face, arms, and hands may pulsate.

Palpation.—The apex-beat is feeble and diffused, with a distinct epigastric pulsation.

Percussion.—Percussion shows an increase in the area of cardiac dullness to the right and upward.

Auscultation.—A systolic murmur is heard in the region of the ensiform cartilage. It is of low pitch, soft, and often faint. The murmur is frequently transmitted slightly to the right. In pure tricuspid regurgitation the pulmonary second sound is not accentuated. The liver often pulsates, and a murmur may be heard in this area.

Diagnosis.—A murmur heard at the ensiform cartilage, systolic in time, and slightly transmitted to the right, not accompanied by accentuation of the second pulmonary sound, with jugular and epigastric pulsation, points to tricuspid regurgitation.

TRICUSPID STENOSIS.

Synonyms.—Tricuspid constriction; tricuspid narrowing.

Etiology.—This is frequently congenital, rarely acquired in origin, and is often associated with malformations of the heart, and accompanied by tricuspid regurgitation.

The prominent symptoms are lividity of the face, dyspnea, vertigo, edema, with scanty and albuminous urine.

Physical Signs.—Upon inspection congestion of the body is prominent. The impulse can be seen in the epigastric region. A *negative venous pulse* is noted in the neck. A thrill may be felt in the tricuspid area. The cardiac area is increased upward and to the right.

A presystolic murmur is heard upon auscultation, at or near the xiphoid cartilage. This may be heard faintly toward the base, but never toward the apex.

Diagnosis.—The diagnosis of this condition can only be made by excluding other valvular lesions.

PULMONARY INCOMPETENCE.

Etiology.—This valve lesion is very rare, and may be the result of injury, or congenital malformations of the heart.

At the second or third interspace to the left of the sternum a loud diastolic murmur is present, which becomes weaker in intensity as the apex of the heart is reached.

Gerhardt has recently called attention to a reduplicated heart-sound which may be heard over the lungs.

PULMONARY STENOSIS.

Etiology.—This condition is extremely rare, and may be due to endocarditis occurring during uterine life. It is almost always a congenital affection. Congenital syphilis has been supposed to be a causative factor.

Pathology.—Fibrous adhesion of the valve segments is most often the cause of pulmonary stenosis.

Symptoms.—Palpitation, dyspnea, cyanosis, and edema are the prominent symptoms when this occurs.

The condition must be diagnosticated by the occurrence of a systolic murmur heard in the pulmonary area. It is not transmitted upward or downward. The second pulmonary sound may be absent or weak. Hypertrophy of the right ventricle accompanies it. The diagnosis must be made by excluding other valvular lesions.

Prognosis of Chronic Valvular Diseases.—So long as compensation remains established, even though the valve lesion be a serious one, the outlook for the patient is good. With signs of failing compensation the slightest valve lesion becomes serious. Age is important, as the condition is most unfavorable in young children. As the valve lesion may go on to progressive changes, repeated attacks of the exciting cause—most often rheumatic fever—may occur.

When valve lesions appear in early adult life, compensation is most apt to become established and remain so for a long time. When valve lesion develops in the aged, the prognosis is good, as few urgent cardiac symptoms are likely to develop.

The male sex does not bear valve lesion as well as the female. Intercurrent diseases render the prognosis unfavorable.

The most serious valve lesion—and for practical purposes the left side of the heart need only be considered—is aortic regurgitation. This is frequently associated with angina pectoris, and, on account of the great hypertrophy of the left ventricle, sudden death from rupture of the heart may take place. Next may be considered aortic stenosis, but this valve lesion is rare, and usually occurs in those past middle life and in old age. Mitral stenosis is next in order, and the least serious of all valvular conditions is mitral regurgitation.

Treatment of Chronic Valvular Diseases.—The treatment of valvular disease should be divided into three periods.

First.—The period of complete compensation, in which no drugs are required.

Second.—The period of failing compensation.

Third.—The period of complete rupture of compensation.

In the first period the patient should, if possible, lead a quiet life free from excitement. The diet should be of a light, nutritious, easily digested character, great care being bestowed upon the condition of the bowels. The patient should be instructed never to strain at stool. Alcohol and tobacco had better be avoided, and if used at all they should be taken sparingly. Exercise is essential, but should not be prolonged to fatigue. Bathing is useful, but the Turkish bath should not be permitted. Sexual indulgence in the majority of cases should be entirely abstained from. The patient should avoid attacks of bronchitis, and if possible should live during the winter in a warm, even climate. It is well, under these conditions, to let some member of the family or an intimate friend know that the patient is suffering from valvular disease of the heart.

The first signs of failing compensation are dyspnea upon exertion, palpitation, and slight edema of the feet;—an examination of the patient will show that hypertrophy and dilatation have been making progress. *Rest in bed is now imperative.* The use of calomel in broken doses, in cases of this sort, is often of advantage. If great signs of dilatation take place, digitalis in some form is a useful drug. It may be given either in the form of an infusion or of the tincture, or digitalin may be used. Strychnia in small doses is also beneficial in this condition.

In aortic insufficiency, when there is great vascular tension, nitroglycerin is often the best drug. If dyspnea is marked, rest in bed should be advised. Morphin, cautiously administered in small doses, often gives great relief. If there be much fluid in the pleura, aspiration may be resorted to. Dropsy should be treated by the administration of calomel, salines, and digitalis. Hot-air baths, or pilocarpin, in this condition, are not advisable. Southey's tubes may be used to relieve the edema.

COMBINED VALVULAR LESIONS.

It would be a great mistake to suppose that valvular lesions only occur singly; combinations of any and all the forms described may occur. The diagnosis of such lesions naturally becomes complicated. An important point in the recognition of such conditions is to endeavor to find the primary seat of the cardiac affection. The primary valve lesion should, if possible, be diagnosticated.

Frequently mitral regurgitation and stenosis are combined; on the other hand, aortic regurgitation and stenosis occur together. It is most common for the mitral and aortic segments to be affected together. The next in frequency are combinations of mitral and tricuspid lesions, and then aortic, mitral, and tricuspid lesions.

Aortic regurgitation and stenosis are more often encountered than mitral regurgitation and stenosis.

EFFECTS OF VALVULAR DISEASE.

When compensation is maintained, very little disturbance manifests itself in other organs, but from long-continued valvular disease, especially after repeated attacks of failing compensation, organic lesions result in other organs. This is brought about by the failure of blood pressure, being decreased in the arterial tree, and a backward pressure manifesting itself in the venous radicals.

The lungs, liver, kidneys, spleen, stomach, intestines, brain and heart-muscle suffer most. When failing compensation occurs, edema first manifests itself in the dependent parts of the body, usually appearing about the ankles, then gradually extending up the limbs, in the arms, and may become general (anasarca). Atrophy of the heart-muscle, accompanied by pigmentation, frequently results from long-continued congestion, called brown atrophy of the heart-muscle. The heart-muscle in this condition becomes indurated and of a yellowish brown color.

As a result of high blood tension in the pulmonary circulation, brown induration of the lungs develops; they become indurated, the blood-vessels dilated, and there is pigmentation in the interstitial substance and alveolar epithelium. Edema, congestion, pulmonary apoplexy, bronchitis, and pneumonia are not infrequent complications.

The symptoms resulting from the foregoing conditions are usually manifested in cough, dyspnea, or orthopnea, and occasionally hemoptysis. In valvular heart disease, chronic bronchitis and repeated attacks of acute bronchitis are not uncommon. Edema and congestion of the lungs are commonly late manifestations, occurring usually before death.

Pulmonary tuberculosis not often follows valvular heart disease. The reverse, however, is not true, as endocarditis not infrequently results as a complication of pulmonary phthisis.

The liver, as a result of long-continued congestion, becomes atrophied, the condition being called cyanotic atrophy, or red atrophy. The appearance of the liver in this condition is much altered. It is larger than normal, of a deep purple color, and on section is mottled, revealing what is called the "nutmeg" appearance, or the liver of heart disease.

This mottling is due to the dilated central veins of the lobules, and causes atrophy of some of the surrounding liver-cells and a deposit of pigment in these cells. Around this congested area the liver more or less retains its color, or becomes lighter, due to fatty infiltration. When the tricuspid valve becomes incompetent, pulsation of the liver frequently results, the impulse being transmitted directly through the venous circulation. The area of liver dullness is increased. Fatty infiltration of the liver sometimes exists in chronic valvular disease.

The kidneys also show similar alterations as a result of the change in blood pressure, the condition being called cyanotic induration. The organ becomes large and indurated, of a deep purple color.

The urine shows marked changes, due to cyanotic induration. The total quantity is greatly diminished and it becomes albuminous, and casts and blood make their appearance.

The spleen also becomes enlarged and congested. Infarcts are common in the spleen and liver.

The stomach and small intestines become congested, and the mucous membrane reveals catarrhal inflammation. As a result of this, digestive disturbances manifest themselves, which are also somewhat influenced by the changes occurring in the liver. The appetite begins to be impaired; there is pain after eating, and constipation frequently occurs. The small intestines also show similar changes. Hemorrhage into both the stomach and kidney are often encountered.

The brain, from failure of compensation, becomes edema-

tous, fluid collects in the subarachnoid and ventricular spaces, and the veins become distended. As tricuspid regurgitation develops, occurring usually from dilatation of the right heart, pulsation becomes apparent in the veins, this being most marked in the veins of the neck.

The dropsy of heart disease begins around the ankles or in the pretibial space, and gradually extends upward. The external genital organs frequently show marked edema, causing obstruction in the urethra as a result of pressure. This often produces annoying symptoms. Pain becomes marked, and inflammation not infrequently results.

HYPERTROPHY OF THE HEART.

Definition.—An increase in the thickness of the walls of the heart, accompanied by increased functional activity with or without alteration of the capacity of its chambers. It may be limited to a single chamber, or to one side, or may affect the entire heart. Hypertrophy of the left ventricle is the commonest of these conditions. Next in order of frequency is hypertrophy of the left auricle, then the right ventricle, and lastly the right auricle.

This occurs in two forms, simple hypertrophy, and hypertrophy with dilatation, called "eccentric" hypertrophy.

Simple hypertrophy consists in an increase in the amount of the heart muscle, its cavities remaining unaltered.

Hypertrophy with dilatation consists in an increase in the heart muscle combined with an increase in the capacity of its chambers.

Etiology.—Hypertrophy of the heart occurs in all conditions in which great muscular effort is required, so that the heart must perform more work.

The factors giving rise to hypertrophy of the heart may be divided into—

First.—Causes relating to the blood-vessels.
Second.—Causes relating to the heart.
Third.—Causes relating to the nervous system.
Fourth.—Toxic causes.

Causes Relating to the Blood-vessels.—If interference occurs with the flow of blood through the small arteries, the blood pressure rises, and cardiac contractions are increased in force to overcome the obstacle. This may be due to in-

elasticity and loss of contractile power in the arteries, to narrowing of their caliber, or to actual obliteration, thus bringing about a marked increase in peripheral resistance. These causes lead to hypertrophy of the left ventricle. When they occur in the pulmonary circuit the right ventricle becomes hypertrophied.

General arterial sclerosis is the chief factor in causing increased peripheral resistance. In this condition the walls of the small arteries become stiff and rigid and are unable to dilate. When the vascular system is filled, the blood pressure necessarily rises, and the ventricles make greater efforts to overcome the resistance, the aortic cusps close tightly, and hypertrophy results. This is common in gout and chronic interstitial nephritis. Extensive atheromatous change in the larger arteries also leads to hypertrophy.

Emphysema and chronic interstitial pneumonia are the principal causes of hypertrophy of the right ventricle. A pleural effusion, by compressing the capillaries, may cause the pulmonary capillaries to become obliterated, and the increased pressure may arise from this cause. Aneurysm also causes hypertrophy.

Causes Relating to the Heart.—Aortic regurgitation and aortic stenosis produce great hypertrophy of the left ventricle, for reasons which have already been explained. (See Aortic Regurgitation and Stenosis.) Mitral regurgitation also gives rise to hypertrophy of the left ventricle on account of the increased amount of blood thrown into the ventricle at the auricular systole. Stenosis of the mitral orifice gives rise to hypertrophy of the left auricle. Hypertrophy of the right ventricle is often due to mitral disease. These lesions increase the blood pressure in the pulmonary vessels.

Active physical exertion continued through a long period of time produces hypertrophy. It is commonly met with in athletes, soldiers, and laborers whose vocations require prolonged physical effort.

Causes Relating to the Nervous System.—Cardiac hypertrophy is met with in those addicted to excessive venery, and is a common occurrence in exophthalmic goiter.

Toxic Causes.—The principal among these are alcohol, coffee, tea, tobacco, and lead.

Pathology.—The heart is increased in size so that in extreme cases it may weigh 600 grams or even more. In hypertrophy of the left side the heart increases in length,

whereas in hypertrophy of the right side the apex is more rounded and the transverse measurement is increased.

Microscopically, the fibers are increased in numbers, and are thickened, the nuclei being swollen. Some degree of myocarditis is almost always associated.

A certain amount of dilatation is usually found with it.

Symptoms.—Among the early symptoms of hypertrophy of the heart is shortness of breath upon exertion. There may be flushing of the face, noises in the ears, and flashes of light before the eyes. The carotids may throb forcibly. There are disagreeable sensations in the epigastrium after taking a full meal. Pain does not occur, as a rule. Headache and vertigo are common, and the patient becomes conscious of the action of the heart especially upon retiring to bed and resting upon the pillow. The pulse is full and strong and of high tension. These symptoms may all continue until changes take place in the myocardium and dilatation occurs. Cerebral hemorrhage from rupture of one of the smaller arteries of the brain may be a complication.

Physical Signs.—It is impossible to make a diagnosis of hypertrophy of the heart without a physical examination.

Inspection.—On inspection there may be bulging in the precordial region. This, however, is not frequent, and is most often the result of previous disease, such as pericarditis. In children, bulging in the precordium is of frequent occurrence in hypertrophy of the heart. The apex-beat is displaced downward, and may be found as low as the eighth intercostal space in the anterior axillary line. Displacement to the right may also be noted. The impulse is heaving and powerful in character. Violent throbbing of the superficial arteries commonly shows itself.

Palpation.—Palpation confirms inspection.

Percussion.—Percussion of the heart is very unsatisfactory and yields questionable results.

Auscultation.—Auscultation gives a booming, prolonged, dull first sound. If the tension be high, the second sound will also be sharp and ringing. Occasionally there may be reduplication of the second sound from absence of synchronous closure of the semilunar valves.

Diagnosis.—The diagnosis rests upon the displacement of the apex-beat usually downward and to the left, the heaving, forcible impulse, with the symptoms of headache, vertigo, gastro-intestinal disturbances, etc.

Prognosis.—The prognosis in uncomplicated cases may be favorable, the danger being due to subsequent dilatation. With proper treatment, chiefly regulation of the habits, excessive myocardial changes may be warded off for years. The younger the patient the more likely it is that hypertrophy may be arrested.

Treatment.—The treatment consists, wherever possible, in the removal of the cause. The bodily functions should be carefully looked after; the patient's diet must be regulated, overeating forbidden, alcohol, coffee, and tea prohibited, and proper rest prescribed. Drugs, as a rule, are not indicated; aconite in small doses is perhaps the most valuable in certain cases.

DILATATION OF THE HEART.

Definition.—Cardiac dilatation, from a pathologic standpoint, may be divided into simple dilatation and dilatation with hypertrophy.

Simple dilatation consists in an increase in the size of the cavity accompanied by thinning of the heart-wall.

Dilatation with hypertrophy consists in an increase in the size of the cavity with increase in the heart muscle.

Simple dilatation is always primary and acute; dilatation with hypertrophy, secondary and chronic.

ACUTE OR SIMPLE DILATATION.

Definition.—*Acute dilatation* is a sudden overdistention of a cavity or cavities of the heart, characterized by serious and often fatal symptoms.

Etiology.—Any condition which produces increased intracardiac pressure may produce dilatation. This may result from severe muscular exercise, forced marches, mountain-climbing, etc. Under such circumstances the right ventricle will suffer most.

After the rupture of a cardiac valve, acute dilatation may take place. The infectious diseases predispose to this condition in causing degenerative changes in the heart-wall.

Sudden death sometimes occurs in diphtheria, enteric fever, etc., where the patient sits up in bed. This is caused by the abrupt distention of the ventricles, followed by paralysis. In croupous pneumonia, overdistention of the right heart takes place and often causes death.

Thrombosis of the pulmonary artery has led to the same result. Sudden plunging into cold water is sometimes followed by acute dilatation.

Pathology.—The cavity affected is distended with dark, partly coagulated blood, and its walls thinned. The right ventricle is more often affected than the left; also the right auricles more often than the left. If obstruction occurs in the systemic vessels, the left ventricle suffers most.

Symptoms.—The symptoms come on abruptly. Dyspnea is a marked symptom. There may be pain in the precordial region; vertigo or dizziness. Nausea and vomiting are not always present, but they frequently appear. Pallor of the face with cyanosis, especially of the lips, is characteristic. Flashes of light appear before the eyes, and the patient may become unconscious. These symptoms may be transitory, or increase in severity so that a fatal result may ensue.

Physical Signs.—A feeble, diffused impulse, with an increased area of cardiac dullness, with weak, rapid, indistinct heart-sounds, are the common physical signs.

Diagnosis.—This depends upon the occurrence of the symptoms and physical signs just described.

Prognosis.—If death does not occur at once, the subsequent changes may lead to it. If the dilatation be moderate, and compensatory hypertrophy take place, recovery may follow.

Treatment.—Absolute rest in bed is important. Cardiac stimulants, such as alcohol, the nitrites, especially nitroglycerin, and hypodermics of strychnin are useful. An ice-bag over the heart may give relief. If improvement takes place, digitalis may be given with benefit. In acute dilatation venesection is of great value. During convalescence tonics should be prescribed.

CHRONIC DILATATION.

Etiology.—The exciting cause is increased intracardiac pressure. When this is gradual, as in valvular lesions of the heart, the dilatation is slow, and is usually compensated for by hypertrophy of the affected cavity. The coronary arteries sooner or later show atheromatous changes in their walls, so that nutrition of the myocardium becomes impaired.

Pathology.—More than one cavity is usually affected, and the heart is larger than normal. The cavities may contain

uncoagulated blood, and the auricles large clots. The auriculo-ventricular ring is stretched on both sides, and the tips of the valve segments do not come together. This may in rare cases be prevented by calcification at the bases of the mitral valves.

The papillary muscles and chordæ tendinæ are shortened so that with the ventricular systole the relative incompetence is increased. The wall of the dilated chamber is pale, and the coronary arteries are diseased and may be partly occluded.

Congestion of the lungs, liver, kidneys, spleen, and intestines accompany cases of long standing.

Symptoms.—As dilatation is slow and progressive, the symptoms may remain latent for a long time. The first signs may be due to imperfect aeration of the blood in the lungs, the brain receiving a deficient amount of arterial blood, the abdominal viscera becoming congested and their functions perverted. The stagnation of the blood current permits transudation of the liquid elements from the blood, and dropsy takes place. Cardiac palpitation is one of the earliest symptoms. It is often accompanied by pain and distress in the precordial area. The rhythm of the heart becomes disturbed, and the patient becomes conscious of it. The pulse is rapid, irregular, and often intermittent. Dyspnea is a prominent symptom.

Respiration is hurried and shallow, and often accompanied by cough and expectoration, which may contain blood. From changes which take place in the stomach and intestines dyspeptic symptoms occur.

The kidneys fail to excrete the normal amount of urine, which is of a darker color and of high specific gravity, containing albumin, often red blood-cells, and casts. The edema which accompanies this condition begins in the feet, and may extend over the entire body until there is a condition of anasarca. The serous cavities are filled with fluid; this is especially likely to happen toward the close of life.

Physical Signs.—Inspection.—The apex-beat is feeble, displaced toward the right, with a diffused impulse, which often presents a wavy character.

Palpation.—Palpation confirms inspection, and the impulse may be so feeble that it can not be felt.

Percussion.—Percussion may show an increase of cardiac dullness laterally, especially to the right.

Auscultation.—The first sound of the heart is feeble, due to the loss of its muscular element, but the valvular sound may be pronounced. The first sound is short, sharp, and feeble, and resembles the normal second sound. It may be intermittent. Reduplication of the first sound may occur, due to a want of synchronism of the left and right ventricle.

Murmurs may be present, due to stretching of the auriculo-ventricular ring, or if murmurs were present previous to the dilatation they lose their intensity or may disappear, this being the result of the weakened heart muscle.

Diagnosis.—The diagnosis depends upon the feebleness of the impulse, its wavy character, the feeble first sound, which is often reduplicated, and the symptoms just enumerated, with signs of edema and perhaps anasarca.

Prognosis.—The prognosis is unfavorable.

Treatment.—The treatment should be directed to the maintenance of the nutrition of the body and the control of the cardiac action. Food must be given in small quantities, at frequent intervals, and it is important that it be highly nutritious. Fatigue should be avoided. It is necessary for the functions of the body to be kept in normal working order. The kidney may be stimulated by diuretics, and a mild laxative be given for the bowels. Digitalis and strychnin are the best drugs for this condition. Basham's mixture is often of use.

DISEASES OF THE MYOCARDIUM.

FATTY INFILTRATION.

Synonyms.—Fatty overgrowth; cor adiposum.

This is a condition in which fat is deposited under the visceral layer of the pericardium, especially marked about the auriculoventricular groove. It also deposits itself between the muscle-fibers, causing some atrophy, and occasionally fatty degeneration may accompany the condition.

The cause of fatty infiltration may be said to be sedentary habits, overeating, and hereditary. Most commonly it is encountered in obesity, the heart being one of the organs which is affected in this general process.

Symptoms and Signs.—The symptoms are rarely subjective, but on examination it will be found that the apex-beat is very faint, or invisible, and auscultation reveals a weak first sound and a relatively distant second sound. This results

from the increase of fat, which interferes with the heart's motion. It has been said that fatal termination from impairment of the contractile power has resulted.

PARENCHYMATOUS DEGENERATION.

This condition is usually associated with infectious diseases and fevers. It is also a forerunner of fatty degeneration. The heart, as a result of this condition, becomes softer, paler, and more friable. Microscopic examination reveals swelling of the heart muscle, and a granular precipitate.

FATTY DEGENERATION.

Etiology.—This may be either partial or general.

It may result from prolonged infectious diseases, following parenchymatous degeneration of the myocardium, and often accompanying this condition. Anemic states, particularly progressive pernicious anemia, cause the affection. Poisons, such as phosphorus, antimony, and arsenic, produce it.

Interference with the circulation of the coronary arteries, due to atheroma, thrombosis, and embolism, usually causes partial fatty degeneration.

Pathology.—The heart, as a rule, is smaller than normal. Its consistency is lax and flabby, and of a yellowish brown color, certain areas being lighter, of a yellowish-gray color. This discoloration (brindled, striated, or tabby-cat appearance) is most marked about the columnæ carneæ and papillary muscles, but it may be quite extensive. The left ventricle is effected more often than the right. The heart muscle is found to be anemic.

Microscopic examination reveals small fatty droplets in the heart muscle, many of the nuclei being completely obliterated and replaced by fat globules. These globules are small, and scattered throughout the muscle-fiber. This fatty process is more marked in certain areas than in others, giving rise to the brindled or striated appearance, the lighter areas corresponding to the fatty degeneration.

Fatty degeneration of other organs frequently accompanies the lesion of the heart, especially when it is due to some general malnutrition.

Symptoms and Signs.—Unless the degenerative process reaches a certain grade, the diagnosis is impossible. Marked symptoms and signs only occur in advanced disease. The

most important of these changes consists in passive dilatation of the ventricles so that the apex-beat becomes weak, diffuse, and even entirely absent. The heart-sounds are correspondingly weak, but clear, and in a high grade of dilatation may be accompanied by a systolic murmur. Enlargement of the liver, due to passive congestion, and albuminuria, may occur in consequence. The radial pulse, on account of the insufficient work performed by the heart muscle, is weak, and in the majority of cases slow, and twenty beats a minute have been recorded. In consequence of this slowing of the heart's action syncope and even apoplexy may occur.

Cheyne-Stokes respiration takes place in grave cases. Among the symptoms may be mentioned dyspnea, especially upon exertion, attacks of asthma (cardiac), palpitation, angina pectoris, and constipation. In the eye, an arcus senilis may be seen.

Diagnosis.—The diagnosis depends upon the history of the case, preceding disease, especially occurring in acute infections in young persons. The slow pulse, the signs of passive dilatation of the ventricles with accompanying symptoms.

Prognosis.—The prognosis is unfavorable as to cure, although the patient may live many years.

Treatment.—The treatment consists in careful regulation of the diet, attention to the functions of the body, and the systematic use of such drugs as alcohol and strychnin. Iodid of potassium and arsenic are recommended by many authorities.

ACUTE MYOCARDITIS.

This is an inflammation of the heart muscle. It usually results from an infected embolus in the course of pyemia. It sometimes arises during diphtheria, typhoid fever, gonorrhea, rheumatic fever, anthrax, and scarlet fever, or it may result by extension from acute endocarditis and pericarditis.

Acute myocarditis usually terminates in abscess formation. However, there may be simply an infiltration of round cells and leukocytes with accompanying blood-vessel changes. The muscle-fibers around this area usually show some degree of parenchymatous degeneration.

The wall of the left ventricle is generally the seat of the condition. The right side is rarely affected. Abscesses, when multiple, are usually pyemic in origin.

The condition may terminate in rupture or aneurysm.

CHRONIC INTERSTITIAL MYOCARDITIS.

Synonyms.—Chronic myocarditis; fibroid heart.

Etiology.—Traumatism, injuries, cold, overexertion, alcohol, tobacco, lead-poisoning, gout, diabetes mellitus, or the infectious diseases, such as malaria, syphilis, and rheumatic fever, may give rise to this condition. Changes in the coronary arteries are causative factors. Chronic interstitial nephritis frequently accompanies this condition, and the general senile changes are responsible, commonly called senile myocarditis.

Occasionally the acute condition may become chronic and so give rise to the disease. It is more common in men than in women, and occurs more frequently after the fortieth year.

Pathology.—This is essentially a cirrhosis of the heart. It frequently involves the wall of the left ventricle, especially about the apex. Hypertrophy is often associated. The fibrous connective tissue surrounds the bundles of heart muscle, causing atrophy. Sclerosis of the coronary arteries frequently accompanies this condition, and sometimes occlusion of these vessels is met with. Aneurysm and dilatation of the heart may develop as a result of chronic interstitial myocarditis. Chronic endocarditis and pericarditis are often associated diseases.

Symptoms and Signs.—These may be entirely latent and the condition in mild grades not likely to be recognized. In the advanced stages pain in the precordium, especially upon slight exertion, becomes prominent. It radiates and shoots down the left arm, and tingling may take place in the fingers. Shortness of breath is a prominent symptom.

The apex-beat is weak and diffused. The pulse is feeble and often intermittent. Marked arrhythmia occurs. Occasionally sudden death takes place and the autopsy may fail to reveal the cause. Constipation and gastric disturbances are prominent. If there be passive congestion, cyanosis and edema of the skin occur.

AMYLOID DISEASE.

Amyloid or wax-like disease of the myocardium results from chronic malaria, syphilis, long continued suppuration,—especially of bone,—and lead-poisoning. By some this is considered to be a degeneration, by others an infiltration.

The amyloid material deposits itself in the interstitial tissue and around the blood-vessels. It is usually accom-

panied by similar changes in other organs, such as the kidneys, liver, and spleen, and its presence can only be suspected by the disease existing in other organs.

Diagnosis of Myocarditis in General.—The diagnosis of myocarditis depends upon a recognition of the cause, the age of the patient, the slow intermittent arrhythmical pulse, with the signs of passive congestion.

Prognosis.—The prognosis as to cure is unfavorable. With proper care and systematic treatment the patient may live for many years.

Treatment.—The treatment consists in proper hygiene, attention to the excretions, easily digested food, and the use of alcohol, strychnin, iodid of potassium, or arsenic in proper doses. Digitalis in this condition is contraindicated. The Nauheim bath treatment is valuable in this disease.

ANEURYSM OF THE HEART.

Etiology.—Aneurysm of the heart most frequently affects the wall of the left ventricle, sometimes the septum between the ventricles, and rarely the right side. This condition is very uncommon. It results from lesions of the muscular fibers of the heart, such as fatty degeneration, acute suppurative myocarditis, and chronic fibrous myocarditis.

Any effort requiring great muscular strain by causing decreased resistance, with an increase in the vascular tension, may give rise to aneurysm. Age is an important factor, this condition usually occurring after forty. It is more frequent in men than in women.

Symptoms and Signs.—The symptoms in this disease are not characteristic.

The diagnosis must be made by exclusion rather than by direct symptoms and signs. Pain in the precordium is usually present. Murmurs, if they occur, are not distinctive, and are usually dependent upon some other cause. If the aneurysm is very large and situated anteriorly, bulging of the chest wall with pulsation is present. It may terminate in rupture. Pericarditis is almost invariably associated with aneurysm of the heart.

Cardiac aneurysm can not be positively diagnosticated.

Prognosis.—The prognosis is hopeless.

Treatment.—The treatment consists in relieving the pain by the use of opiates.

RUPTURE OF THE HEART.

Rupture of the heart usually results from over-exertion, the heart muscle being, as a rule, previously diseased, either from fatty infiltration, or more frequently from fatty degeneration, acute suppurative myocarditis, or previous aneurysmal dilatation. The rupture may cause a sudden gush of blood into the pericardial sac, or a slow leakage may continue for a long time.

Disease of the valves, especially of the aortic valves, on account of great hypertrophy of the left ventricle, may give rise to rupture.

Rupture most frequently occurs in the anterior wall of the left ventricle. Disease of the coronary arteries is often the cause of the diseased myocardium. Softening of the heart muscles, due to new growths and echinococci, has caused rupture of the heart.

Symptoms and Signs.—If the disease comes on gradually, with slow leakage, the symptoms are not diagnostic. There may be marked dyspnea, with palpitation and pain, partial suppression of the urine, or even anuria due to the lowering of blood pressure. Where the rupture takes place suddenly, great pain and precordial distress occur, rapidly followed by death.

The physical signs in either condition may show a weak or absent apex-beat. The sounds are feeble and irregular. An increased area of cardiac dullness, due to the effusion of blood into the pericardial sac, may occur.

Prognosis.—The prognosis is absolutely unfavorable.

Treatment.—The treatment consists in applying ice-bags to the heart, and the liberal administration of opium.

NEW GROWTHS AND PARASITES.

All malignant growths, carcinoma, as well as sarcoma, have been found in the heart. When they are secondary, they occur particularly in the pericardium and endocardium rather than in the muscular substance. New growths are extremely rare. Of nonmalignant growths there are fibroma, lymphoma, and myoma. Hydatid disease of the heart has been noted.

Diagnosis.—A diagnosis is impossible except, perhaps, by exclusion.

Symptoms.—The symptoms of pericarditis are almost invariably present.

Prognosis.—The prognosis in any condition is unfavorable.
Treatment.—The treatment is purely symptomatic.

NEUROSES OF THE HEART.

By a neurosis is meant a disturbance in action, or function, independent of an organic lesion.

ARRHYTHMIA.

Definition.—An irregular rhythm of the heart's action.
Etiology.—This may be due to either direct, reflex, or toxic causes, or to any combination of these.
Direct Causes.—Any disturbance of the pneumogastric nerve or of a brain lesion would constitute a direct cause, such as apoplexy, brain tumor, abscess of the brain, pressure upon a nerve trunk by enlarged glands or a neoplasm.

It often occurs in endo- and pericarditis, myocarditis, and arteriosclerosis. Alterations in the blood itself—anemia, chlorosis, etc.—may give rise to the condition.

A distention of the stomach by gases, causing pressure upward, may produce arrhythmia. In some forms of Bright's disease it commonly occurs.

Reflex Causes.—The condition may be due to an organ in the body away from the heart, especially found in disease of the abdominal organs. It also arises from shock, trauma, and from pain.

Toxic Causes.—The infectious diseases; digitalis, muscarin, coffee, tea, tobacco, and alcohol.

Symptoms.—As the name implies, the principal symptoms consist in irregularity in the heart's action. Intermissions, with difference in the volume of the pulse, take place. The heart-beats may occur at long or short intervals, and may be bounding or feeble.

Prognosis.—This depends upon the cause and the ability to remove the productive factor.

Treatment.—The treatment is symptomatic.

PALPITATION.

By palpitation is meant heart-beating which is perceptible and annoying to the patient. It may be due to causes within the heart itself as organic disease, and exophthalmic goiter.

Reflex Causes.—Reflex causes may give rise to it—disease

of the stomach, disease of the genito-urinary apparatus, and sexual excesses.

Toxic causes consist in the abuse of alcohol, tobacco, tea, and coffee. Gout and anemia are productive factors, and the condition occurs in inanition and marasmus.

Symptoms.—Perceptible heart's action, which varies in intensity, is the important factor. The attacks depend on slight causes, and may occur at night, awakening the patient from sound sleep. Associated symptoms may be pallor of the face, with cold, clammy sweat, sighing respirations; also vertigo, tremor, cyanosis, and syncope may occur. The attack may last from a few minutes to the greater part of the day.

Prognosis depends upon the cause; usually favorable.

Treatment.—Treatment is symptomatic. Rest in the recumbent posture is important.

IRRITABLE HEART.

Two varieties of this condition exist: (1) A form occurring in young persons; (2) the so-called soldier's heart.

Etiology.—Overeating, tobacco, alcohol, hard study, and nonsystematic exercise are potent causes in its development. In soldiers it is often due to forced marches, especially in those persons not accustomed to taking exercise. The male sex is oftener affected than the female.

Symptoms and Signs.—These consist in uneasiness about the heart, irregularity of the cardiac action, occasionally slow, then rapid beats, and perhaps slight pain. The first sound is often muffled ("murmurish"). There are no distinct signs of hypertrophy, although if the condition be prolonged this organic change may occur. There are disturbances of digestion, eructation of gases, and constipation. The person is irritable (nervous), and in great anxiety in regard to the condition of his heart, the irregularity of which is apparent to him.

Prognosis.—With proper care, the prognosis is favorable, although the condition may lead to organic change.

Treatment.—Regulation of the habits and avoidance of tobacco, alcohol, tea, and coffee. Muscular exertion must be systematic and guarded. Change of scene and pleasant surroundings are often beneficial. Small doses of the bromids are useful, but digitalis should be avoided unless organic change (dilatation) takes place.

TACHYCARDIA.

By this is meant increased frequency of the heart's action. The condition may be permanent, periodic, or paroxysmal. It is permanent in exophthalmic goiter. In the fevers and during convalescence from fevers, from great and rapid blood loss, and in anemia it appears periodically. It is spoken of as paroxysmal when it occurs from fright or some nervous irritation.

Symptoms.—The symptoms consist in an increased frequency of the pulse, occurring in paroxysms, which may be unprovoked, or excited by a trivial cause. The pulse rate may be increased to 140 or as high as 200 beats per minute, rarely more. Paroxysmal tachycardia usually sets in suddenly in persons who are apparently in good health. The heart shows no evidence of organic lesion in the paroxysmal cases.

Prognosis.—The prognosis is favorable as to life, but the condition is extremely obstinate, and usually continues until there follows degeneration of the heart.

Treatment.—The treatment should consist in the relief of the attack. The use of drugs is exceedingly unsatisfactory. Rest, in the recumbent posture, is perhaps the most useful agent in procuring relief. Hydrotherapy is useful in many instances.

BRADYCARDIA OR BRACHYCARDIA.

Bradycardia is the slow action of the heart. The normal pulse range is from 60 to 80 per minute, but in bradycardia the pulse may fall to 40, 30, or even less per minute. As in the opposite condition (tachycardia), bradycardia is permanent, temporary, or paroxysmal.

It is usually permanent in organic disease of the brain.

Temporary bradycardia is much more common, occurring in some of the infectious fevers, especially yellow fever, and in puerperal fever. It may occasionally occur in diphtheria. The accumulation of bile in the blood slows the pulse. The absorption of toxins in the alimentary canal may have a similar effect.

Paroxysmal bradycardia sets in abruptly in persons seemingly in good health, and without apparent cause.

Symptoms.—The symptoms consist in a slowing of the pulse rate. If the pulse becomes markedly slow, the patient may lapse into a semicomatose condition, with a pallid or flushed face, and with cool or hot extremities.

Prognosis.—The disease is commonly regarded serious, as the principal causes of the affection are diseases of the brain and spinal cord.

Treatment.—Stimulants, such as digitalis and nitroglycerin, are not indicated so long as the heart supplies the wants of the body. Oil of camphor, hypodermically, is of use in this affection. Strychnin should also be given as a general heart tonic.

ANGINA PECTORIS.

Definition.—Angina pectoris is a neurosis of the heart, commonly due to sclerosis or atheroma of the coronary arteries or myocardial disease, characterized by severe pain in the region of the heart, radiating to the left shoulder and left arm, and other symptoms.

Etiology.—Occlusion of the coronary arteries is an important factor, as is also sclerosis. Predisposing causes are age and sex; the disease occurs after the middle period of life, and in an overwhelming number of cases in the male sex.

Heredity, in so far as it gives rise to sclerosis, may have some slight predisposition. Syphilis is an important predisposing factor. Sedentary habits, and lack of proper exercise, have also been noted as predisposing causes.

Pseudo-angina Pectoris.—Pseudo-angina pectoris occurs particularly in younger subjects, and in women in connection with hysteria and neurasthenia.

Symptoms.—The attack takes place suddenly. The patient, probably without prodromes, is seized with severe lancinating pain in the precordial region, which radiates to the left shoulder and down the left arm, so that the arm and fingers are frequently numb. The attack may be so severe as to cause death at once. The patient describes the sensation as a feeling of compression of the heart, and as though it were impossible for him to draw his breath. The heart's action during the attack is variable; the circulation may be entirely unaffected, or there may be palpitation and arrhythmia. The pulse may be decreased or increased in frequency. The disease occurs commonly in combination with aortic affections, especially stenosis of the aortic valves. It is often found in chronic myocarditis.

The duration of the attack varies from half a minute to several hours. The intervals between the attacks also vary. Exceptionally, the patient may have but one attack, from

which he may recover. It is, however, usual for the patient to have several, the disease proving fatal in one of them.

Symptoms of Pseudo-angina Pectoris.—The disease occurs with pain in the precordium in young persons, particularly women, in association with hysteria and neurasthenia. The pain may radiate down the left arm. During the attack the patient may pass a large quantity of urine, or there may be eructation of gas.

Diagnosis.—The diagnosis is easy as a rule; the excruciating pain, the anxiety of the patient, the signs of arteriosclerosis, and the occurrence in the male sex, all point prominently to angina pectoris.

In pseudo-angina pectoris the accompanying phenomena of hysteria or neurasthenia are important diagnostic points.

Differential Diagnosis.—

Angina Pectoris.	*Pseudo-angina Pectoris.*
Male sex after forty years of age.	Young females of neurotic temperament.
Some disease of heart or blood vessels.	No organic disease of the heart.
Attacks not so frequent and more prolonged.	Attacks more frequent, and briefer in duration.
Often loss of consciousness.	Rarely loss of consciousness.
Often fatal.	Rarely fatal.

Prognosis.—The prognosis in true angina is always unfavorable. Cure frequently takes place in pseudo-angina.

Treatment.—The treatment must be divided into two parts: the treatment of the attack, and the care of the patient in the interval. In the attack, hypodermics of morphia are necessary. Inhalation of amyl nitrite, or the employment of nitroglycerin in full doses may prove beneficial.

In the interval between the attacks the patient must lead a quiet life. The functions of the body should be well looked after, and doses of nitrite of sodium with strychnin are of use.

Pseudo-angina may be relieved by Hoffmann's anodyne, or applications of hot or cold water over the heart.

DISEASES OF THE ARTERIES.

INFILTRATIONS AND DEGENERATIONS.

Calcareous Infiltration.—Calcareous infiltration frequently affects the arteries, either in the course of atheromatous changes, or the artery may simply be impregnated with lime-salts without any foregoing pathologic change, being converted into a stone-like tube. This infiltration also occurs in the final organization of thrombi.

Amyloid Disease.—Amyloid infiltration of the arteries results from syphilis, chronic malaria, tuberculosis, suppuration—especially that of bone—and chronic lead-poisoning. The smaller (medium sized) arteries are almost without exception the ones which are affected by this infiltration. This may lead to a weakening of the arterial wall, and aneurysm may result. Amyloid infiltration in the internal organs usually accompanies this condition.

Fatty Degeneration.—Simple fatty degeneration of the intima usually occurs in individuals beyond middle life, and Virchow has described a similar condition occurring in chlorotic girls. Fatty degeneration, however, is usually one of the stages in atheroma.

Hyaline Degeneration.—Hyaline degeneration may also attack the arteries, commonly involving the intima.

ARTERITIS, OR INFLAMMATION OF THE ARTERIES.

ACUTE ARTERITIS.

This condition usually results from some infective process. It is frequently encountered in pyemia, sometimes in ulcerative endocarditis and enteric fever. It may affect any of the arteries. The changes noted are distention of the vasa vasorum, the intima becomes roughened, and there is an inflammatory infiltration into one or more coats of the arteries which may go on to the formation of pus, or in some instances an aneurysm develops. Acute aortitis is rare, being a condition similar to ulcerative endocarditis. Thrombosis may result from roughening of the intima.

Micro-organisms may be found upon the surface of the projection or vegetation, and in the inflammatory area in the coats of the arteries.

Symptoms.—Acute arteritis may occur during the process of a primary lesion or during convalescence. The important symptom is spontaneous, localized pain in the region affected, most frequently a limb, exaggerated by movement and by pressure. Upon the occurrence of thrombosis a painful cord may be felt in the affected area. The pulse is obliterated, there is numbness and tingling with anesthesia, coldness of the skin, and swelling. The local temperature is lowered, and gangrene may follow.

ARTERIOSCLEROSIS.

Synonyms.—Arteriocapillary fibrosis; Gull and Sutton's disease.

Definition.—A sclerosis of the arteries, affecting usually the intima, but the adventitia and media may be involved. This may be local or general.

Etiology.—Arteriosclerosis is more common in the male than in the female sex.

This condition usually results from advancing age, but the rule has many exceptions, as the young are frequently attacked. It is a well-known saying that, "A man is as old as his arteries."

Heredity seems to play an important part in the causation, as some families show a distinct tendency to arteriosclerosis early in life. Improper living, sedentary occupations, over-eating, overwork, occupations which give rise to severe muscular exertion, and syphilis are all important factors in the causation of this disease. Persons suffering from gout show a marked tendency to arteriosclerosis. Alcohol and chronic lead-poisoning are also important causes.

Chronic interstitial nephritis gives rise to this condition; however, kidney disease may be secondary to arteriosclerosis, and again, both conditions, no doubt, often occur simultaneously from a common cause.

Pathology.—Arteriosclerosis may be diffuse, affecting many of the small arteries of the body, those of the brain, of the heart, and of the extremities. The arteries of the lungs are less commonly involved. Again, the sclerosis may be confined to the larger arteries, such as the aorta.

1. Atheromatous Arteritis.

This disease is primarily one of the intima. Early there is a milky opacity of this coat, and a thickening takes place in this membrane. A yellowish spot may next appear in the thickened area, which is the result of fatty degeneration. This fat may be carried away by the blood stream, leaving an ulceration, or the fatty patch may be converted into a calcareous plate. If the former result, the artery is predisposed to aneurysmal dilatation. In the latter event, emboli may be thrown into the circulation, providing the atheromatous plates are dislodged. This atheromatous change most frequently involves the aorta, but the iliacs, femorals, and arteries of the limbs and the cerebrals are often involved.

Microscopically, the thickening simply consists in the formation of fibrous connective tissue. As a result of this, the elasticity of the vessel becomes impaired and may dilate and form an aneurysm.

2. Arteritis Obliterans.

This condition, as a rule, occurs from syphilis. Occasionally, this change has been found in the cirrhotic kidney, fibroid lung, and in blood-vessels after ligation. The intima shows great thickening as a result of fibrous connective-tissue formation. The elastic coat and the muscular coat show little change, and the adventitia may in some instances be thickened. In consequence of this, the lumen of the artery becomes diminished, and in some instances completely obliterated, often leading to gangrene of the parts supplied. The intima does not show a tendency to fatty degeneration, so that it does not predispose to aneurysm, and the condition is usually associated with other signs of constitutional syphilis, and affects most frequently the small arteries.

3. Diffuse Arteriosclerosis.

This condition is wide-spread, occurring frequently during middle life. The intima shows marked thickening, and the disease affects, more or less, the aorta and its branches.

As a consequence of sclerosis, the vessel-walls become inelastic, the onward flow of the blood is somewhat prevented, as the resistance is increased, and sooner or later the left ventricle becomes hypertrophied. This is especially so when it is associated with chronic interstitial nephritis. The arterial tension becomes high, and the diseased arteries are liable to aneurysmal dilatation. As a result of roughening of the intima, thrombosis not infrequently occurs.

Many degenerative changes may appear from narrowing of the blood-vessels, such as softening of the brain, fatty degeneration of the heart, etc.

Symptoms.—Great changes may take place in the arteries without giving rise to symptoms. The condition is easily recognizable if the external arteries are the ones affected; thus, the hardening in the radial and temporal arteries may be felt, but the internal arteries are not open to scrutiny. The principal symptoms depend upon the high-tension pulse, which is full and strong and difficult to obliterate. Next in importance are changes which relate to hypertrophy of the heart, particularly the left ventricle. The apex-beat is dislocated downward and to the left, the impulse being forcible and heaving; the second aortic sound is clear, ringing, and accentuated. The involvement of the coronary arteries may develop symptoms of angina pectoris. If there be changes in the cerebral vessels, paralysis of various kinds may result. The ophthalmoscope may reveal changes in the retinal vessels.

Vertigo is a prominent and frequent symptom. Urinary symptoms occur in a majority of the cases. The urine is increased in amount, of low specific gravity, rarely containing casts or albumin, pointing strongly to contracted kidney, which often is associated. If dilatation follow hypertrophy of the heart, relative insufficiency develops. This may be difficult to diagnosticate from actual organic valvular defects. The urine under such a condition will have changed, being lessened in amount, and of high specific gravity, containing albumin and casts (due to congestion of the kidney). Edema may result, as in failing compensation from valvular disease. Respiratory symptoms occasionally appear, such as relate to bronchitis and allied conditions.

Diagnosis.—This depends upon the tortuous temporal arteries, hard, whip-like radials, high-tension pulse, accentuated second aortic sound, and hypertrophy of the left ventricle.

When the atheromatous condition involves the internal arteries, the diagnosis is more difficult, and the accentuation of the second aortic sound and hypertrophy of the left ventricle are less distinct.

Prognosis.—The prognosis varies in individual cases. The patient may live for some time but the changes in the arteries are never amenable to cure.

Treatment.—The patient's life should be carefully regulated, excesses avoided in food and drink, and there should be no exertion of any description, mental quietude being insisted upon. Alcohol should be absolutely prohibited.

If the history of syphilis occur in the case, a prolonged treatment by iodid of potassium is of use. In other cases, the best results are obtained by giving the nitrites, such as the nitrite of sodium or nitroglycerin. The bowels should be carefully regulated.

ANEURYSM.

Definition.—"An aneurysm is a circumscribed tumor, containing fluid or solid blood, communicating directly with a canal of an artery, and limited by a tunic which is called the sac" (Hilton Fagge).

Etiology.—Trauma is an important etiologic factor. Any condition which weakens the wall of a blood-vessel may give rise to aneurysm; thus, arteriosclerosis is an important cause. Any condition which raises local blood pressure may be causative, such as muscular effort, heavy lifting, wrestling, sudden fright, straining at stool, parturition, etc. Syphilis is important; many authors estimate as high as eighty per cent. of all cases due to this cause. Alcohol, in giving rise to arteriosclerosis, may be mentioned as a causative factor. Gout and lead-poisoning act in the same manner. The greatest number of cases occur between the ages of thirty and forty, and aneurysm is more frequent in the male than in the female.

General Considerations.—Dilatation may occur in any artery in the body, so that an aneurysm may vary in size from the so-called miliary aneurysm to an immense tumor. The condition is more frequent in some countries than in others; thus, it is comparatively rare in Germany, France, and Italy, but more frequent in England. The greater number of aneurysms in this country occur in foreigners. The majority of aneurysms appear in the thoracic aorta, next in the abdominal aorta, the subclavian artery, and the innominate artery. In the aorta itself, the arch seems to be affected in the greatest proportion of cases.

External aneurysms belong particularly to the domain of surgery, internal medicine having to do chiefly with aneurysm as it occurs in the aorta and its main divisions in the chest and abdomen, and the miliary aneurysm, particularly of the brain.

In the brain the middle cerebral artery is affected most frequently.

Pathology.—*Varieties.*—Aneurysms may be true or false. A true aneurysm is a circumscribed dilatation of one or more coats of an artery. A false aneurysm has for its wall the surrounding tissues, the blood-vessel having ruptured. Aneurysm may also be classified as regards its shape; when oval or spindle it is called *fusiform*, or *cylindric aneurysm.* When one portion of an artery is dilated into a pouch-like formation it is called a *saccular aneurysm;* when the blood finds its way between the coats of an artery, as a result of rupture of the inner coat (the middle coat may also be ruptured), it is called a *dissecting aneurysm.*

As has before been stated, the usual cause of aneurysm is arteriosclerosis. The blood-vessel loses its elasticity, the vessel becomes weakened, and any sudden strain may cause it to give way.

Miliary aneurysms are sometimes produced as a result of destruction of the outer coats of a vessel; for example, in a tuberculous lung, the process involving the larger blood-vessels. The outer coat becomes diseased and the inner protrudes, forming what is called miliary aneurysm. Again, an aneurysm may become very large, in some instances reaching an enormous size. The aneurysmal sac may contain laminated clots, often being healed. In very old aneurysms this clot may assume the appearance of being fibrous. In many aneurysms, however, no coagula are to be found. Atrophy, from pressure of surrounding tissues, commonly accompanies aneurysm. The vertebras, the ribs, and the sternum are not infrequently involved, and portions of these structures may entirely disappear. Pressure upon the bronchi may cause bronchiectasis, and pressure on the lung, atelectasis.

Rupture may result in the form of a slow leak or rapid gush. When the first portion of the arch of the aorta is involved the rupture may occur into the pericardium (see Hemopericardium). The rupture of the thoracic aorta may take place externally, into the pleural sac, mediastinum, bronchi, trachea, lungs, and esophagus. Rupture into the superior vena cava has been reported. External rupture is not uncommon.

Aneurysm of the abdominal aorta may cause atrophy of the vertebras and surrounding structures. Rupture may take place externally or into the peritoneal cavity. Aneurysms of the brain may be of quite large size or of the miliary character.

Hypertrophy of the heart, as a rule, occurs, being chiefly due to the arteriosclerosis which usually precedes the development of aneurysm.

Symptoms.—The symptoms may be latent for a long period. The early diagnosis of aneurysm is often impossible, the symptoms being misleading. Among the earliest and most important symptoms is pain. It may occasionally be absent, but in the majority of cases it is the first, most important and enduring symptom. It differs greatly in continuity, variability, and character. It may be slight, or, on the other hand, severe enough to threaten life, or disturb the comfort and rest of the person affected. It is usually acute and paroxysmal, subject to remissions and exacerbations, or it may be dull, gnawing, and localized in the position in which the aneurysm occurs. Paroxysmal pain in some region in close relation to the aorta is always suggestive of aneurysm. The pain is often worse at night. In abdominal aneurysm the pain is likely to be severe and shoot through to the spine. It is intense and wearying in character, and rarely absent. The heart may remain entirely free from signs or symptoms for some time. However, in a number of cases palpitation occurs.

Important symptoms are pressure phenomena; thus, the pain may often be due to the aneurysmal sac pressing upon a nerve-trunk. Occasionally there is hyperesthesia and anesthesia. Hoarseness and aphonia may result from pressure upon the recurrent laryngeal nerve. Unilateral sweating and change in the size of the pupils are often symptoms of pressure upon the sympathetics. From pressure, particularly upon the left bronchus, tracheal tugging occurs. Pressure upon the pneumogastric may cause vomiting, and upon the esophagus may give rise to dysphagia. As a result of pressure upon the thoracic duct there may be extreme emaciation. Pressure upon the bronchus may result in dyspnea, which may be paroxysmal in character.

Cough and hemoptysis are frequent symptoms in aneurysm of the aorta. Pressure upon the superior vena cava may give rise to distention of the veins of the neck and face, causing cyanosis with edema. Pressure upon the inferior vena cava gives rise to similar conditions of the limbs and congestion of the viscera.

Changes in the pulse may occur, especially if the aneurysm is situated in the ascending part of the aorta; the pulse, com-

pared with the apex-beat, is retarded. In the ascending aorta the carotid pulse may also be delayed. The location of the aneurysm may cause a delayed pulse of one side; for example, the left carotid and subclavian pulse may follow those of the right side.

Physical Signs of Thoracic Aneurysm. — Inspection.— If the aneurysm have eroded the ribs or sternum, protruding itself externally, a pulsating tumor is seen, often producing a blue or livid discoloration of the skin.

Palpation.—Palpation may show a downward displacement in the apex-beat of the heart, due to pressure from above downward and to the left, without signs of marked hypertrophy. The aneurysm, if palpable, reveals an expansile pulsating tumor, and a more or less distinct thrill, which is systolic in time. Tracheal tugging may also be noticed upon palpation. A distinct diastolic shock may sometimes be present over the base of the heart.

Percussion.—Percussion elicits flatness over the tumor.

Auscultation.—Auscultation gives a distinct systolic bruit from the rush of blood through the distended tumor.

Signs of Rupture.—Rupture of an aneurysm is recognized by the instant collapse of the tumor, and symptoms of profuse and rapid hemorrhage.

Diagnosis.—The diagnosis depends upon the recognition of the etiologic factor; the symptoms of pain, palpitation, and dyspnea, presence of tumor, with expansile pulsation, thrill, and bruit, and often dislocation of the apex-beat of the heart, accompanied by pressure phenomena.

Prognosis.—The prognosis is always grave, although cure may take place.

Treatment.—Absolute rest in bed is of the utmost importance. A diet in which fluids are largely eliminated (Tufnell treatment) is of use in some cases. The symptomatic treatment consists in the administration of large doses of iodid of potassium. The use of opium for the relief of pain should be delayed as long as possible.

Surgical Treatment.—The surgical treatment may consist in ligation, filipuncture, needling, electropuncture, compression and wiring, wiring with electrolysis, and the hypodermic use of gelatin.

ANEURYSM OF THE ABDOMINAL AORTA.

Etiology.—The same causes which give rise to aneurysm of the thoracic aorta produce aneurysm of the abdominal aorta. The pressure symptoms relate more to the abdominal viscera and the lower extremity. The pain is constant, gnawing, shooting through to the back, and may radiate down the legs. The physical signs are the same as those in thoracic aneurysm, although the tumor is not nearly so likely to make its appearance.

Diagnosis.—Occasionally, masses of various kinds, such as tumors of the left lobe of the liver, of the stomach, or of the pancreas, or large glands, may give rise to some of the physical signs of aneurysm. However, the expansile pulsation is absent, although this is sometimes difficult to determine. If, then, the patient be placed in the knee-elbow position, and if the tumor falls forward, the signs of aneurysm will disappear.

Prognosis.—The prognosis is practically the same as that of thoracic aneurysm.

Treatment.—The treatment before described for thoracic aneurysm should also be adopted in cases of aneurysm of the abdominal aorta.

PART III.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE NOSE.

ACUTE RHINITIS.

Definition.—An acute catarrhal inflammation of the nasal mucous membrane, characterized by copious secretion of a serous or mucous character.

Synonyms.—Acute coryza; acute nasal catarrh.

Etiology.—The affection is most often caused by cold and exposure, although it may result from trauma and the inhalation of irritants.

Pathology.—In the first stage there is hyperemia and slight swelling of the mucous membrane and of the turbinated bones, accompanied by dryness of the membrane. This is followed by a profuse serous or mucoserous discharge, which later may become purulent.

Symptoms.—The attack may begin with slight chilliness and malaise, with some headache and pain referred to the nasal and frontal bones. The temperature is subfebrile, and the pulse is not altered. Stenosis of one or both nasal chambers may occur, due to the swelling of the mucous membrane of the nose. This is followed by profuse discharge of a mucous, serous, and finally seropurulent character. Sneezing is common, and the conjunctivæ may be injected. The discharge is sometimes acrid, and the lips and anterior parts of the nose may be slightly excoriated.

Complications.—The inflammatory process may invade the frontal sinus, the antrum of Highmore, the Eustachian tube, and the nasopharynx.

Prognosis.—Uncomplicated cases recover in from five to

seven days. It must be remembered that the disease may aggravate a preexisting nasal or pharyngeal condition.

Treatment.—Prophylaxis consists in the avoidance of cold; and persons who are subject to nasal catarrh should undergo a hardening process by means of hydrotherapy, etc. Mild alkaline nasal washes and a weak solution of cocain applied to the nose gives temporary relief. Where there is great secretion, small doses of atropia or tincture of belladonna are useful.

CHRONIC RHINITIS.

Definition.—A chronic catarrh of the nasal mucous membrane, frequently implicating the nasopharynx and giving rise to hypertrophy, especially of the turbinated bones.

Synonyms.—Chronic nasal catarrh; postnasal catarrh.

Description.—There are several more or less well-defined varieties of chronic nasal catarrh. (1) *Simple chronic catarrh*, in which there is irritability of the mucous membrane, especially of the septum and turbinated bones. There is clogging of one or both nostrils, and the patient has a special liability to *catch cold.* The nasal secretion is thick and tenacious.

(2) *Hypertrophic rhinitis* is characterized by hypertrophy of the turbinated bones, causing a partial or complete stenosis. Adenoid vegetations are apt to occur in this form. From the stenosis of the nasal passages the affected person becomes a "mouth-breather," and this condition is apt to be aggravated at night.

When this disease occurs in young children, and especially when complicated by adenoids, the facies become dull and stupid, and deformities of the chest are apt to develop.

(3) *Atrophic rhinitis* may be the result of the hypertrophic variety. On account of the foul odor which comes from the nose, the condition has been called *ozena* and *coryza fœtida.* Ozena is a name given to any foul discharge from the nose, and may be a symptom of syphilis, glanders, foreign bodies in the nose, necrosis, etc.

The disease occurs more often in women than in men, and especially in the early periods of life. Upon inspection the mucous membrane in this variety is thin and covered with grayish white crusts, which on removal leave an excoriated surface, but very rarely a true ulcer. The symptoms are loss of smell, even to the offensive odor which comes from the nose.

Treatment.—Thorough cleansing of the nasal surfaces is important; reduction of growths and hypertrophied tissue gives relief. The condition had better be treated by a specialist.

DISEASES OF THE LARYNX.

ACUTE LARYNGITIS.

Definition.—An acute catarrhal inflammation of the larynx.

Etiology.—Sudden changes in temperature, accompanied by moisture, the inhalation of irritant gases, the excessive use of the voice in shouting, speaking, or singing may produce the condition. It occurs commonly in some of the acute infectious diseases, such as influenza, measles, whooping-cough, etc., and in the various diatheses, such as the gouty, rheumatic, tuberculous, and syphilitic.

Pathology.—The mucous membrane usually presents a mucous exudate with some injection of the surrounding blood-vessels, the entire membrane being somewhat swollen. Ulcers and vesicles have been noted in this condition.

Symptoms.—The chief symptoms are soreness or pain in the larynx, with a dry, irritating cough, and some degree of hoarseness. Later, small patches of mucus streaked with blood may be expectorated. Occasionally aphonia may take place. There may be slight fever and general malaise. Dyspnea may occur in children.

Diagnosis.—The laryngoscope reveals an acute inflammation of the larynx.

Prognosis.—The prognosis is favorable.

Treatment.—In severe cases the patient should be kept in bed and the atmosphere of the room moistened. The patient should refrain from talking. An ice bandage around the neck gives great comfort. Inhalations of steam, medicated by compound tincture of benzoin and paregoric, will allay cough and irritation. In children, if dyspnea becomes marked, emetics should be used.

CHRONIC LARYNGITIS.

Definition.—Chronic catarrhal inflammation of the larynx.

Etiology.—All causes which produce acute laryngitis may give rise to the chronic form. This condition frequently fol-

lows acute or subacute attacks. Interference with nasal respiration has been given as a potent cause.

Pathology.—The mucous membrane usually shows some thickening, and may be granular. Later in the course of the disease atrophic changes and the formation of new fibrous tissue may result.

Symptoms.—The symptoms are hoarseness, and sometimes complete loss of voice. Cough is not a constant symptom, but if present it may be either dry or productive. If there be abundant expectoration, the trachea and bronchi will be found to be involved. Constitutional symptoms are, practically, always absent. The laryngoscope may show the appearance of the vocal cords to be dull, and grayish or pinkish in color, both cords being most often affected.

Diagnosis.—The diagnosis depends upon the etiology, and the exclusion of tuberculosis and malignant diseases.

Treatment.—The treatment should consist in complete rest of the voice. The general health should be carefully looked after. Local applications to the larynx, and inhalations, are of some use. Strychnin to improve the muscular tone is of value. Electricity has also been used.

EDEMA OF THE LARYNX.

Definition.—This condition is not an independent affection, but occurs as a complication in various diseases. The disease is commonly called edema of the glottis. The glottis, however, being a space, can not become edematous, and the term is a misnomer. Two varieties are described, primary and secondary, or passive edema.

Etiology.—Simple edema may arise from trauma, such as the swallowing of a hard body and applications of medicines to the larynx. A condition may occur in the larynx, similar to that which takes place in the skin, known as angioneurotic edema. This usually appears during young adult life, and most frequently in women. Edema of the larynx may arise from the administration of some drugs—for example, iodid of potassium. Primary edema may also arise from infectious conditions, such as the entrance of micro-organisms into the larynx. It occurs in some of the infectious diseases, such as enteric fever, diphtheria, hydrophobia, etc.

Secondary edema may be due to tuberculosis, syphilis, carcinoma, and may occur from some of the infectious diseases,

such as influenza, smallpox, etc. It may be due to the inflammation of a deep cervical gland. The general causes are those which give rise to dropsy, such as disease of the heart, kidneys, lungs, etc., or from pressure from new growths of the mediastinum, etc.

Symptoms.—The patient complains of pain in swallowing, and a feeling as if there were a foreign body in the throat. The voice is thick and muffled, or there may be aphonia. As the glottis does not close completely, the patient is likely to choke on taking food into the mouth and swallowing, as particles of the ingested substances may get into the larynx. Respiration becomes difficult and orthopnea may take place. If death is threatened, the patient presents the symptoms of suffocation. The laryngoscope shows the mucous membrane to be tense, pale, and swollen.

Prognosis.—General edema is a serious condition. The prognosis depends upon the cause. If the condition be due to sepsis it is almost invariably fatal.

Treatment.—The treatment is that of dropsy occurring in other parts of the body. Surgical treatment, such as scarification, intubation, tracheotomy, etc., is often necessary.

LARYNGISMUS STRIDULUS.

Synonym.—Spasm of the larynx.

A disease limited to neurotic individuals, occurring in both children and adults.

SPASM OF THE LARYNX IN CHILDREN.

Rickety children are subject to this affection, in whom it often appears as a reflex disturbance from teething, indigestion, intestinal parasites, exposure to cold, catarrh of the air-passages, irritation of the prepuce, and cerebral or cerebrospinal disease. It is most common in infants under the age of two.

Symptoms.—The disease comes on abruptly, the child being attacked most often at night with shortness of breath, followed by closure of the glottis, which remains closed sometimes for twenty or thirty seconds. The face during this period becomes anxious, and cyanosis is a prominent feature. This is followed by relaxation of the spasm, giving rise to a high-pitched inspiration. The spasm may repeat itself several times and then subside, returning after a day or two. These recurrences may continue for a period of several weeks, with

gradual improvement. The disease may, however, prove fatal, the child choking to death. Convulsions occur if the attack be very severe.

Prognosis.—The disease may prove fatal, particularly in boys.

Treatment.—Any constriction about the neck must be speedily loosened, and as much fresh air admitted to the room as possible. The feet may be immersed in a hot mustard bath, cold compresses being applied to the head and chest; and sinapisms to the nape of the neck are useful. Hypodermics of morphin and atropin may be given in severe cases. Chloral, by the bowel, is occasionally very useful. Compression of the phrenic nerve with the index finger placed between the two lower attachments of the sternocleidomastoid muscle, frequently repeated, has been successful. Intubation and tracheotomy is necessary when the attack is severe and convulsions are threatened. Emetics are also useful in this condition. Tonic treatment, such as cod-liver oil, syrup of the iodid of iron, etc., should be given. Good hygiene is of importance.

SPASM OF THE LARYNX IN ADULTS.

The condition is usually reflex, due to some underlying disease. It is never fatal. It may be due to the entrance of a foreign body into the larynx, or to a laryngeal tumor, and the ulceration from syphilis or tuberculosis may give rise to it.

Symptoms.—The attack comes on at night with similar symptoms to those occurring in children, but the recurrences are not so frequent nor so severe.

Prognosis.—The prognosis is favorable.

Treatment.—Antispasmodics are of use. The spray of a 4% solution of cocain, or antipyrin administered by the mouth usually promptly overcomes the spasm. Severe measures are rarely ever necessary.

CROUP.

Synonyms.—Spasmodic croup; pseudodiphtheria.

Etiology.—This affection is probably due to some infectious agent which has not yet been discovered. It frequently follows sudden chilling of the body, the attacks occurring particularly in children before the age of puberty. Heredity plays some part as an etiologic factor. The condition consists of a catarrhal inflammation with slight exudate.

Symptoms.—The attack begins suddenly, most often at night. The first indication is usually a hoarse, metallic cough, followed by dyspnea. The dyspnea may become extreme, and the child gasp for breath. Cyanosis of the face and extremities occurs in extreme cases. The temperature may be elevated from 102° F. to 104° F. The cough is usually unproductive, but toward the close of the attack free expectoration may take place. Albuminuria may occur as in other infectious diseases.

Diagnosis.—The disease may be mistaken for diphtheria, but a bacteriologic examination will show the presence of the Klebs-Löffler bacillus in diphtheria.

Prognosis.—Favorable.

Treatment.—The child should be at once immersed in a warm bath, and an emetic promptly given. Syrup of ipecac or turbeth mineral may be used for this purpose. Between the attacks the child should have tonic treatment of cod-liver oil, syrup of the iodid of iron, or compound syrup of the hypophosphites.

DISEASES OF THE BRONCHI.

BRONCHITIS.

This disease is divided into two varieties, acute and chronic bronchitis.

SIMPLE ACUTE BRONCHITIS.

Synonyms.—Acute catarrhal bronchitis; acute bronchial catarrh and acute trachitis.

Definition.—Acute bronchitis is an inflammation of any part of the bronchial mucous membrane, excepting the terminal bronchial tubes.

Etiology.—This disease occurs at any age. Sudden changes in the weather, especially damp and cold weather, give rise to this affection. Very hot rooms and bad ventilation and wetting of the feet are causative factors. Inhalation of noxious gases and chemicals may produce the disease. It occurs in many of the acute infectious diseases, such as enteric fever, influenza, and whooping-cough; also in many chronic affections of the heart, particularly in valvular disease characterized by failing compensation. It is also found in chronic renal disease, diabetes, gout, and in chronic diseases

of the spinal cord. Occupations which give rise to exposure and the vicissitudes of the weather, such as the coachman, laborer, etc., are important predisposing factors. The administration of some drugs, particularly iodid of potassium, may cause acute bronchitis.

Pathology.—By many the affection is believed to be of microbic origin. The mucous membrane of the trachea and larger bronchial tubes, and sometimes the smaller tubes, are affected. When the irritant lodges upon the mucous membrane the first change produced is a granular degeneration of the epithelial cells and congestion of the part, followed by desquamation of the epithelium, later infiltration of the submucosa with leukocytes and swelling of the mucous glands. The mucous surface is dry, and it appears injected. Later a muco or mucopurulent exudate may be found upon the surface. Some of the bronchial tubes may be more or less completely filled with this exudate, which often collects at the bifurcation of many of the bronchi as a result of the stream of expired air. In this manner small masses of exudate are expectorated.

Microscopic examination of the expectoration reveals the following: it usually contains many epithelial cells, various micro-organisms, leukocytes, and sometimes red blood-cells, depending upon the severity of the lesion. The disease may be localized to almost any area, such as the trachea,—being called trachitis,—or one or more bronchial tubes, and if the condition extends into the smaller bronchial tubes and alveoli, bronchopneumonia results.

Symptoms.—Acute bronchitis usually begins with pain of moderate intensity in the chest, the pain being referred to the region under the sternum. There may also be muscular pain, especially of the intercostal muscles. Cough is an early symptom; it may be due to the accompanying laryngitis, but more frequently to irritation of the mucous membrane of the trachea and larger bronchi. The intensity of the cough varies in different cases. It is at first unproductive, or a very small patch of glairy mucus may be brought up, later becoming mucoid or mucopurulent. Occasionally the expectoration may be tinged with blood. This occurs particularly in cachetic individuals and in those of an alcoholic temperament. Dyspnea is moderate. There may be slight fever, the temperature ranging from 100° to 102° F. The appetite is lost, the patient generally being uncomfortable. In infants, catarrh of

the nose and throat usually precedes the attack of bronchitis, the respirations being increased in frequency, the nursing child rejecting the nipple and pausing before resuming to nurse. This is due to the fact that the mucous membrane of the nose is affected, the child being unable to respire while the nipple is in its mouth. The temperature in the bronchitis of infancy rarely exceeds 102° F. or 103° F. Should the temperature rise above this, the physician should be upon the alert, as it is most likely that the finer tubes have become implicated and a bronchopneumonia has developed. In ordinary cases the attack lasts about a week, the expectorations becoming looser after the fourth or fifth day. In subacute forms of the disease the symptoms may be less marked, and the duration of the affection longer.

Physical Signs.—If the process affect the upper part of the trachea alone, the laryngoscope may reveal a pinkish or red appearance of the mucous membrane, and physical signs may be entirely absent.

Inspection.—The inspection is negative.

Palpation.—The palpation is negative, giving the signs of normal lung structure.

Percussion.—Normal pulmonary resonance is produced upon percussion, the exudate in the bronchial tubes not being sufficient to obscure the note.

Auscultation.—Upon auscultation, dry rales are at first heard, followed later by large and small moist rales. These are heard both anteriorly and posteriorly over the entire chest.

If the inflammatory process affects the trachea and the largest bronchi, no rales may be present at all, as the exudate in the first stage is not great enough to materially lessen the lumen of the tubes.

Diagnosis.—The diagnosis depends upon the history of exposure, the substernal pain, the dry cough, followed by an expectoration of a muco or mucopurulent character, with the occurrence of dry and moist rales.

Treatment.—The prophylaxis consists in the avoidance of injurious influences and a hardening process should be instituted by means of hydrotherapy in the young, especially those who are predisposed. The temperature of the room should be between 68° F. and 70° F. In the first stage steam inhalations are of use. Mustard plasters and turpentine stupes, applied to the chest, are often beneficial. For the cough, opium or its derivatives are best. Expectorants should be

given when the cough becomes looser. In adults, heroin in $\frac{1}{16}$ of a grain doses acts well.

FIBRINOUS BRONCHITIS.

Synonyms.—Plastic bronchitis ; croupous bronchitis.

Etiology.—This is a rare disease, occurring after inhalation of noxious gases such as ammonia, and steam. It may be acute, but is usually chronic. It has been observed during the course of erysipelas, scarlet fever, enteric fever, and other diseases.

The exudate which appears occasionally in the bronchial tubes during an attack of diphtheria should be regarded as diphtheria, and not as ordinary fibrinous bronchitis.

Pathology.—The larger or smaller tubes may be affected, the fibrinous exudate being formed upon the surface of the mucous membrane of the bronchi. The exudate varies in firmness. The expectorated material will be found to contain casts of the bronchial tubes. These are easily recognized by allowing the sputum to be placed in water and teased apart.

The exudate is found to consist chiefly of fibrin. Some of the epithelial lining may adhere to this ; leukocytes and occasionally red blood-cells are also found, as well as Charcot-Leyden crystals, and eosinophilic cells.

Symptoms (of the Acute Form).—The acute form is very rare, and cases may occur in children. Prodromes may take place, such as slight headache, cough, and malaise. A chill, however, may usher in the attack. It is followed by dyspnea, cough, scanty expectoration, and severe substernal pain. The dyspnea increases in severity and may give rise to symptoms of suffocation. The cough becomes severe and paroxysmal, and may have the hoarse tone of laryngeal croup. The sputum, at first, is not characteristic, and may contain slight amounts of blood. Rarely, fibrinous fragments are brought up in the early stages of the disease. Later, entire casts of the bronchi may come away with a violent expulsive effort, with free hemoptysis, relief of all the symptoms following at once. With ensuing recovery the symptoms abate. If this favorable event does not occur, the disease may prove fatal in from a few days to a few weeks.

Symptoms (of the Chronic Form).—In the chronic form the symptoms may come on after a more or less prolonged attack of bronchitis, although the disease may begin abruptly

as in the acute form. The symptoms are the same as those recorded in the acute form, and they may assume all grades of severity.

Recovery is, however, the rule, although the general health of the patient may be greatly impaired by the frequency and severity of the successive attacks. The signs of emphysema and atelectasis or consolidation may occur in the chronic variety, although in the uncomplicated disease the percussion note is not changed. No rales are characteristic of this condition, but sonorous and sibilant rales commonly occur. Diminished thoracic expansion may be observed on the affected side.

Complications and Sequels.—Tuberculosis, pneumonia, and very frequently compensatory emphysema may develop. If the affected bronchus becomes impervious, atelectasis results.

Diagnosis.—The disease is extremely rare, and can only be diagnosticated by the characteristic sputum. It may be mistaken for laryngeal diphtheria, and a bacteriologic examination of the exudate may be necessary.

Prognosis.—When this disease occurs in the acute form in previously healthy individuals, recovery may take place. The chronic form in cachectic individuals is fatal. A high range of temperature is unfavorable, especially if pulmonary complications occur. Hemoptysis is not necessarily a serious complication.

Treatment.—No agents are known which are of avail in loosening or softening the fibrinous exudate. Inhalations of steam are often useful. In robust individuals emetics may be beneficial. Opiates are necessary when there is severe pain.

CHRONIC BRONCHITIS.

Definition.—Chronic catarrh of the bronchial tubes.

Varieties.—Chronic dry catarrh ; bronchorrhea ; fetid bronchitis.

Etiology.—This is a disease of middle life or advancing years, and is rare in young subjects. It often follows repeated attacks of acute bronchitis, and all the etiologic factors concerned in the acute form are causative in the chronic variety. The disease may assume a chronic type from the beginning ; this occurs especially in older persons, or in those who suffer from constitutional cachexia, gout, renal disease, cardiac disease, or alcoholism. The disease is often symptomatic, accompanying chronic pulmonary tuberculosis, emphysema, and

asthma. In chronic valvular lesions, especially mitral disease, in which pulmonary congestion occurs, chronic bronchitis is likely to be present.

Pathology.—The bronchi, bilaterally, are usually affected, and only in rare instances are local lesions found. Such a condition may be the result of tuberculosis.

The bronchi may show a variety of lesions; the mucous membrane may be greatly thickened, marked proliferation of the epithelial cells and desquamation being found. Profuse muco or mucopurulent exudation may be present, or the bronchi may show atrophic changes. The mucous membrane is thinned, the character of the epithelium changed, and in some instances the epithelium is absent. Sclerotic changes may be present, and contraction results in narrowing of the lumen, or bronchiectatic changes may follow. Emphysema commonly follows chronic bronchitis. The expectoration may vary, in some instances being profuse and watery, or it may be mucopurulent and, rarely, completely absent. The expectorated material contains epithelium, often being modified; also leukocytes, rarely red blood-cells, Charcot-Leyden crystals, and various micro-organisms.

Symptoms.—Cough, expectoration, and disturbance of respiration are the principal symptoms. The cough is variable, and is usually severer early in the morning, when the exudate which has accumulated during the night begins to shift its position, and the patient is compelled to cough until the exudation is dislodged and brought up. The cough may be hacking, dry, or paroxysmal. In the dry variety it is not productive. The sputum may consist of tough masses, resembling coins, of circular form, termed "nummular" expectoration. In many instances it is abundant and purulent, or it may be abundant and thin. The dyspnea occasionally may be quite marked; in other instances it is insignificant. When the bronchitis occurs in cardiac or renal disease the dyspnea is often marked, and is then due to the underlying condition.

Physical Signs.—Inspection.—The inspection is negative, except that dyspnea may be apparent.

Palpation.—Palpation is negative, unless large quantities of fluid be contained in the tubes, when the vocal fremitus may be diminished. If there is but a small quantity of fluid in the large tubes, bronchial fremitus may be felt, known as the "rhonchus."

Percussion. — In uncomplicated cases percussion gives normal pulmonary resonance. If emphysema occurs the signs relating to this condition are present.

Auscultation.—Upon auscultation, all varieties of rales are heard diffused through the chest. Occasionally they may be moist, and sometimes dry. They are usually heard at the lower parts of the lungs posteriorly, due to the extravasation of the fluid to the dependent portions. In these areas the respiratory murmurs may be obscured and be bronchovesicular in character.

Dry Bronchitis (sometimes called Dry Chronic Catarrh).— In this form the expectoration is slight, tough, and tenacious, or may be absent altogether. Upon auscultation, dry rales occur. Emphysema is a complication, and attacks of asthma are common.

Bronchorrhea.—A rare form, characterized by an excessive amount of secretion. The character of the expectoration differs: in some instances it is thin and watery, or it may be transparent and ropy; this variety was termed by Laennec "chronic pituitous catarrh." Under some circumstances the expectoration is of a mucous, semifluid consistence. This variety has been termed "purulent bronchorrhea." The quantity of expectoration varies from one to three pints. The fluid commonly separates into three layers, the upper one composed of a frothy material, the middle one consisting of a clear, thin fluid, and the lower one a viscid, purulent or cellular substance.

The pathology of bronchorrhea is not well understood; however, emphysema is constantly present, due to the bronchial channels being filled with a secretion. Bronchiectasis is also likely to be associated.

Dyspnea and cough are always present, often being persistent and paroxysmal. In the early morning, a spell of coughing and expectoration, which may last for several hours, is frequently followed by comparative freedom for the remaining portion of the day. Asthma often complicates the disease. Anemia and night-sweats have been noted as accompanying symptoms.

Fetid Bronchitis.—In this variety of bronchitis the secretions undergo decomposition and give rise to foul odors. Bronchiectasis is very apt to be present. The disease is usually chronic. It may arise in the course of other diseases. There is great impairment of the general health; fever may develop, and there may be much pain and coughing. It is

easily diagnosticated on account of the foul odor, not only of the expectoration, but of the breath. Bronchopneumonia may develop, and the condition may terminate in gangrene of the lungs. Clubbed fingers and arthropathies may occur.

Prognosis.—The prognosis is guarded.

Treatment.—The treatment consists in meeting the indications as they arise. Disinfectants may be of use; carbolic acid in form of a weak spray and turpentine internally may have some influence upon the fetor. The apartments of the patient must be thoroughly ventilated, and a formalin lamp may be necessary for thorough disinfection.

Course of the Disease.—The course of chronic bronchitis is protracted, remissions and exacerbations occurring, greatly influenced by the season, so that in summer the cough may be at times entirely absent. When the disease has existed for many years, pulmonary emphysema or tubercular infection may result. The right heart dilates, with or without hypertrophy.

Diagnosis.—The diagnosis is not difficult. It is important to determine whether the bronchitis is a primary disease or a secondary manifestation of other diseases, such as those of the heart, kidneys, gout, or cachexias, etc.

Prognosis.—The disease is not amenable to cure. There may be frequent improvements and relapses. Commonly, changes develop, which have just been described, such as emphysema, bronchiectasis, and hypertrophy of the heart.

Treatment.—The patient should be removed to as favorable an atmosphere as possible. Nutritious food in moderate amounts is necessary; it should consist chiefly of fruits, milk, and the cereals. Meat should not be allowed in large amounts. Alcohol is not necessary in the treatment. Inhalations of various substances are of little avail. It may be necessary to reduce the amount of expectoration or control the cough. This is best accomplished by some form of opium. The prominent symptoms will require treatment.

BRONCHIECTASIS.

Definition.—By bronchiectasis is meant a dilatation of a bronchial tube, commonly due to disease of the lungs or bronchi.

Etiology.—This condition may follow measles or pulmonary tuberculosis. In the majority of cases it results from a chronic inflammation of the bronchial walls.

Foreign bodies in the air-passages, and obstruction from pressure by aneurysm, tumors, enlarged glands, and cicatricial contractions in interstitial pneumonia, are factors in the causation.

It sometimes results from an unresolved croupous pneumonia.

Pathology.—The condition may be local or general; the dilatation saccular, cylindric, or irregular. Bronchiectasis, in the broadest sense of the word, results from obstruction, as noted in the etiology.

The dilated bronchial tubes reveal marked changes in the walls of the bronchi. The epithelium may change from the cylindric to the squamous variety; the muscular fibers and elastic tissue show atrophic changes. The cavities are frequently filled with an exudate.

Symptoms.—The cylindric form can not be diagnosticated. In the sacculated variety, when there are large cavities, the symptoms may be suggestive. There is cough, expectoration, which may be of large amounts of a mucopurulent character, sometimes fetid; it may suggest gangrene of the lungs or a foul-smelling empyema which is connected with a bronchus. The dyspnea depends upon the amount of bronchial obstruction; hemoptysis is uncommon, and may be slight or severe. Diarrhea and emaciation may accompany the condition. Emphysema, chronic bronchitis and asthma are often associated with bronchiectasis.

Sputum.—In milder cases there is nothing distinctive about the sputum. Where there are large saccular cavities, the sputum is, however, characteristic. It is abundant, and raised in large amounts after a period of retention. It is thin, purulent, grayish-green in color, and, where decomposition has taken place, there is a foul odor. Upon standing, it separates into three layers: an upper frothy, a middle thin, and a lower thick and granular layer.

Microscopically, it may be composed almost entirely of pus-cells, with more or less fatty epithelial cells, which vary in outline, many being squamous, others cylindric, and forms varying between these. Many micro-organisms are usually present.

Physical Signs.—Over a large bronchiectatic cavity the physical signs will vary, depending upon whether the cavity is filled or empty, superficial or deep-seated.

The percussion note will be dull when the cavity is full, or high-pitched and tympanitic when the cavity has become evacuated after a paroxysm of cough. Deep-seated dilatations

are very difficult to detect by percussion, as the sounds elicited are modified by emphysema, or by induration of intervening lung substance.

The respiratory changes may consist in feeble expansion and diminished murmur, or there may be prolonged expiration with harsh bronchovesicular breathing. Large bronchiectatic cavities, distended with air, may give cavernous or amphoric breath-sounds. There may be either lack of vocal resonance and fremitus, or the vibrations may be increased. When there are large cavities posteriorly which are partly filled with fluid, bubbling rales may occur. Dry and moist rales may also be present, due to associated bronchitis. The physical signs of the associated bronchitis and emphysema may be present and lend additional difficulty to the diagnosis.

Complications and Sequels.—Hemorrhage may take place, due to ulceration of the bronchial wall. Abscesses and induration of the surrounding lung sometimes occur. Pulmonary emphysema, with dilatation of the right heart and the consequent visceral changes, due to venous congestion of the liver, spleen, and kidneys, occur. In prolonged cases, amyloid degeneration may take place. Arthropathies of the long bones, and especially of the terminal phalanges, appear in this disease.

Diagnosis.—The diagnosis in some cases is very easy, and when cavities appear they must be differentiated from tubercular cavities, pulmonary gangrene, and empyema.

Differential Diagnosis.—

Bronchiectatic Cavity.	*Tubercular Cavity.*
Most often situated at the base of the lung, the signs being most prominent posteriorly.	Most often at the apex of the lung.
Sputum characteristic, foul, and abundant. No tubercle bacilli.	Sputum blood-streaked, not foul, and contains tubercle bacilli.
No fever nor sweating, and not great emaciation.	Fever, sweating, and great emaciation.

Prognosis.—The condition is practically incurable and may last for years.

Treatment.—The general health of the patient must be maintained, and inhalations of creasote, turpentine, and eucalyptus, as deodorizing agents, may be useful. Iodid of potassium in large doses has been advised. Surgical interference is justifiable when the physical signs point to an accessible cavity.

BRONCHIAL ASTHMA.

Definition.—A condition characterized by paroxysms of dyspnea, appearing at irregular intervals, with constitutional symptoms.

Synonyms.—Spasmodic asthma; nervous asthma; essential asthma.

Etiology.—Heredity is important, the disease occurring in nervous or neurotic individuals, in whom worry or fright is apt to bring on an attack. It is common in phthisic families, and is likely to appear in persons subject to neuralgic attacks.

It is a disease of the young, although it is infrequent in infancy, appearing more often in males than in females.

The disease may follow attacks of bronchitis, especially of that form taking place in the acute infectious diseases, as measles, whooping-cough, etc. It is commonly associated with chronic bronchitis and emphysema. The inhalation of dust, the pollen of certain plants, fog, fumes, vapors, certain odors that emanate from some animals may produce an attack of asthma.

Reflex causes, as irritation from nasal polypi or other affections of the nasal mucous membrane, and causes relating to the gastro-intestinal tract, skin, or genito-urinary center, may act in a like manner. It is more common in the Hebrew race.

Pathology.—Three principal theories have been entertained as to the origin of this nervous disease: (1) That it is a result of bronchial spasm, and the clinical phenomena and the effects of certain drugs administered appear to support this view; (2) that the symptoms are due to spasm of the diaphragm, but prolonged spasm of the diaphragm would seem to be incompatible with respiration; (3) known as the hyperemic theory, depends upon the irregular, acute swelling of the bronchial mucous membrane, a similar condition to that appearing upon the skin in urticaria.

There is no general accepted theory with regard to the pathology of asthma. It occurs in families of nervous tendencies, and the pathology is not uniform in every case. The form of asthma in cardiac disease is known as cardiac asthma, and the variety in disease of the kidneys, known as renal asthma, must not be confounded with this condition. In heart and kidney disease asthma should always be considered a symptom, and not as a substantive affection.

Symptoms.—Usually the attack begins suddenly, but may be preceded by prodromes, such as a feeling of general uneasiness and discomfort, or abnormal sensations in the larynx, accompanied by coughing, sneezing, coryza, and tenderness in the epigastrium. The attack most often commences at night; the patient awakens with chest pains, great anxiety, and marked dyspnea, which causes him to go to the window for fresh air. The skin becomes pale and cyanotic. The patient is often bathed in cold perspiration. The pulse is increased in frequency. The temperature may be normal or subnormal, except in protracted cases, when from the irritation of the cough there may be slight elevation of temperature.

The respiratory disturbance in asthma is characteristic. The inspiration and expiration are accompanied by high-pitched whistling rales, which may be heard a considerable distance. The accessory muscles of respiration are called into play, the expiration being labored and difficult. The sterno-cleidomastoid and scaleni may be seen contracting upon inspiration.

Dyspnea is rather expiratory than inspiratory, the difficulty being that the patient can not get the air from his lungs. Upon percussion there is a tympanitic resonance over the lungs. Compensatory emphysema is almost certain to develop, and the physical signs of this condition may be made out. Upon auscultation the vesicular murmur is obscured by whistling sounds, these being the dry rales, which at the end of a paroxysm give place to moist rales, when expectoration also appears. In the majority of cases there is during the attack a scanty, tough mucus, followed by a more abundant liquid expectoration toward the end of the attack. Small gelatinous masses are often found in the sputum, known as the perles of Laennec.

Upon microscopic examination, the sputum contains leukocytes, many being eosinophiles, epithelial cells, sometimes oxalate of lime crystals, Charcot-Leyden crystals, and Curschman's spirals. These spirals are supposed to have some relation to the pathology of the affection.

The frequency of the attack is very variable. It may occur nightly for some time, and periods of freedom follow lasting for months or years. Complete recovery is, as a rule, rare after well-developed attacks. In children recovery may be looked for oftener than in adults.

Diagnosis.—The diagnosis is easy, but the symptoms

should be carefully studied, in order, if possible, to determine the underlying causes.

Prognosis.—Usually favorable as regards the attack, but complete recovery is rare. Emphysema and chronic bronchitis are very apt to develop.

Treatment.—Persons who are subject to asthma ought to live as much as possible in the open air. The living- and sleeping-rooms must be well ventilated. A nutritious and easily digested diet should be given, and heavy meals at night forbidden. Warm and proper clothing in winter and in damp weather is essential.

Treatment of the Paroxysms.—Narcotics and antispasmodics are usually employed. Chloral, whiffs of chloroform or ether, amyl nitrite by inhalation, or morphia and atropia hypodermically are useful drugs. Relief sometimes follows the inhalation of nitre paper cigarettes, which also contain lobelia and stramonium. Sinapisms and turpentine stupes to the chest may be of value.

Where the attack has lasted some time emetics may be of some benefit.

Treatment between Paroxysms.—Good hygienic surroundings are necessary. Medicaments which favor nutrition in general, cod-liver oil, iodid of iron, courses of arsenic, the nitrites, and quinin are of especial use.

Change of climate and hydrotherapy are useful in most instances.

HAY-FEVER.

Definition.—Hay-fever is a neurosis characterized by symptoms which chiefly relate to the upper air-passages, often terminating in asthmatic attacks.

Synonyms.—Hay asthma; rose fever; Bostock's catarrh.

Etiology.—Summer season and a neurotic temperament are predisposing factors, and it is said the exciting factor is pollen of flowers. In some cases there is a predisposition to the affection from the fact of preexisting chronic nasal trouble. It often occurs in gouty and lithemic individuals. There is no doubt that in some cases the disease is hereditary.

Pathology.—No changes have been found that are characteristic of hay-fever. In many cases there is vascular swelling of the nostrils, which may be occluded, and there will generally be found a profuse serous exudation. The principal

changes which occur are supposed to be due to vasomotor paralysis.

Symptoms.—The symptoms come on about the same time each year, sometimes at exactly the same hour and day as on the preceding year.

The early symptoms are usually a sensation of uneasiness, such as itching and tickling in the nose and throat, followed by more or less frontal headache, lacrimation, sneezing, coughing, and nasal obstruction. The catarrhal inflammation of the eyes, nose, and throat soon becomes intense, and a profuse watery discharge from the nostrils appears. The conjunctiva is red and painful, and the patient is extremely uncomfortable.

The degree of severity of these symptoms varies in different cases. In some instances they are extremely mild, while in others they are quite severe. Occasionally high fever, headache, and prostration may confine the patient to his bed. At the height of the disease the nasal mucous membrane is swollen, which may often show at the anterior nares.

Sneezing, with a copious flow of mucus from the nose, which may even continue so as to disturb the patient's rest at night, is common. Cough, although not a constant feature, is usual, and may result from an intense bronchitis and last some time after the active paroxysm has subsided. Often attacks of asthma appear. This comes on late, as a rule, and gives rise to symptoms of an asthmatic seizure. Skin eruptions, such as urticaria and herpes, are quite common, and are extremely annoying to the sufferer.

Diagnosis.—The diagnosis is not difficult. The periodicity in relation to season, the acute onset, with the catarrhal symptoms, are so definite that the disease can hardly be mistaken for anything else.

Prognosis.—The prognosis as regards life is favorable, but very little can be done to abort or prevent a return of the attacks.

Treatment.—A change of climate may alone give permanent relief. For the eyes mild washes of boric acid are useful, and the severe nasal symptoms are temporarily relieved by a 4% or 5% solution of cocain. In the gouty cases the treatment by mineral acids is sometimes of decided benefit.

Syrup of hydriodic acid is sometimes useful.

DISEASES OF THE PULMONARY STRUCTURE.

PSEUDOHYPERTROPHIC EMPHYSEMA.

Synonyms.—Vesicular emphysema; pulmonary emphysema.

Two entirely different morbid conditions have been described under this name. Thus, where air escapes into the interstitial tissue, which may be due to rupture of lung substance, the condition is sometimes termed surgical emphysema; and a change associated with overdistention of the alveolar walls, an enlargement of the alveolar spaces of the lungs. The first form occurs in association with diseases in which there are violent paroxysms of coughing, injuries, and surgical interference. The second variety is the common form known as pulmonary emphysema, which will be described.

VESICULAR EMPHYSEMA.

Etiology.—It may result from bronchitis, pertussis, or asthma, due to the violent attacks of coughing which occur in these conditions, and it may appear in individuals who give no such history. It is a common disease in advancing age, and it may take place as a senile change. Occupations requiring prolonged straining of the lungs, as glass-blowers, and blowing of wind instruments, may produce the disease.

Occasionally the disease is met with early in life. Men are more frequently affected than women.

Pathology.—The essential change in the lungs is the loss of elasticity from overdistention of the walls of the air vesicles, producing consequent weakening, with atrophy of the elastic tissue in the alveolar septa. The walls of the alveoli yield more and more to the pressure of the inclosed air, and become permanently distended, and similar processes take place in the adjacent alveoli, often the septa are destroyed, and two or more alveoli communicate. Not only the elastic tissue of the lung, but also the intra-alveolar capillary network is destroyed; and if the process be extensive, great numbers of the air-spaces are enlarged and permanently distended, so that the elasticity of the lung, which is necessary for complete

physiologic respiration, particularly expiration, is permanently reduced.

The process is not always a primary one. It may develop as a result of other lesions, to which it is secondary. If for any cause disease of a certain portion of the lung occur, in which a lobule or lobe becomes incapacitated, the adjacent parts assume additional activity in taking upon themselves more work, the vesicles becoming distended and overexpanded, often causing them to lose their elasticity, and compensatory emphysema results. This compensatory form develops in the upper lobes when disease of the lower lobes occurs, or in one lung when the other lung is extensively diseased. It has also been called vicarious emphysema.

The condition involving the greater part of both lungs, essential pulmonary emphysema or substantive emphysema, is the disease which will now be described.

Upon postmortem examination, as the lungs are removed from the chest they do not collapse; on the contrary, they may bulge out when the incision is made along the edge of the sternum. The alveolar structure is soft, downy, relaxed, inelastic, and pits upon pressure. The lung is gray in color, being almost bloodless. Bladder-like projections, which are composed of overdistended alveoli, are noticed, and collapse upon section.

The upper portions of the lungs seem to be dry, while the dependent parts are moist from edema.

Symptoms.—Cough and expectoration, usually due to the accompanying bronchitis, is present to some extent. Shortness of breath is progressive, occurring first only upon exertion, but later appearing continuously.

Bronchitis, bronchiectasis, dilatation and hypertrophy of the heart, especially of the right ventricle, and kidney disease are apt to be present.

Physical Signs.—Inspection.—The appearance of the chest is characteristic, the inspiratory form in varying degrees being noted. The chest is broad, deep, and short; the shoulders are raised; the upper ribs are close together, and the lower ribs farther apart. The anteroposterior diameter, compared with the normal diameter, is increased, and the costal angle is obtuse. The nipple may be opposite the fifth rib, and the impulse of the heart often be seen in the sixth intercostal space, being pushed downward by the left lung. The impulse may be obscured on account of the overlap-

ping of the lung interposing between the heart and the chest-wall.

Palpation.—Palpation gives diminished vocal fremitus.

Percussion.—The note is vesiculotympanitic or tympanitic. The liver is pushed downward, and liver dullness partly obscured.

Auscultation.—The breath sounds are distant, indistinct, and scarcely audible, expiration being prolonged so that the normal ratio of inspiration and expiration may be reversed. Rales relating to the bronchitis actively associated are also heard all over the chest.

Diagnosis.—The contour of the chest, the prolonged expiration, the feebleness of the breath-sounds, the dyspnea, the cough and history all make the diagnosis easy.

Prognosis.—Recovery—that is to say, restoration to the original condition—does not occur in this disease, although the duration of life is usually prolonged.

Treatment.—Treatment should be directed to the improvement of the general health and observance of proper hygiene. Attacks of bronchitis should be prevented if possible, as they may tend to add to the mechanical dilatation of the lungs from the cough. Where bronchitis is associated with a great deal of expectoration, the terebinthinates and balsams are of use.

Expectorants, such as apomorphia, ipecac, and the ammonium salts, are often of especial value. If the cough becomes oppressive, opium or its derivatives may be used at short intervals with great caution. The iodids and strychnin are of use, and careful attention should be given to the heart, and the first signs of dilatation should be observed. When they occur, digitalis and caffein are of use. Inhalations of compressed air and expiration into rarefied air have been followed by good results in some instances.

PULMONARY ATELECTASIS.

There are two varieties—congenital and acquired.

The *congenital form* occurs at birth, in which the lung of the new-born has never contained air. The *acquired form* occurs in the course of many diseases, being secondary.

The lung is airless and collapsed. The lung tissue may not be altered at first, being merely a mechanical compression. After a certain time, however, inflammatory changes take

place. The lung tissue assumes the condition to which the term "splenization" has been applied, on account of its resemblance to splenic tissue. In the more advanced stages the congestion gives place to a dry state, which is spoken of as "carnification." A change also noted in advanced stages is the formation of fibrous connective tissue. The involved area sinks when placed in water, and does not crepitate.

ATELECTASIS OF THE NEW-BORN.

This condition is due to imperfect respiration. The lung prior to birth is in a state of atelectasis, being an airless structure. If at birth an insufficient quantity of air enters the lungs, they remain uninflated (atelectatic), and at the autopsy no inflammatory change is noticed, so that the lungs may be expanded by the blowpipe.

ACQUIRED ATELECTASIS.

This may occur in two ways—first, by plugging of the bronchus or bronchiole by means of a fibrinous exudate. This happens especially in the narrow bronchi of young children, so that no air enters the related vesicular structure upon inspiration, causing the vesicular structure to be cut off. The air in the area beyond the plugged bronchus disappears by absorption. Collapse of the vesicles takes place, giving rise to a circumscribed atelectasis. This condition is commonly found in the lungs of children dying from bronchopneumonia, especially if this be secondary to measles, whooping-cough or diphtheria. Weakened respiratory effort, besides the plugging of the bronchus, is an important factor in bringing about this change. The second cause is in compression of the lung, such as may result from pleural effusion, hydrothorax, and pneumothorax, where the lung tissue is compressed and forced together with resulting atelectasis. This is by far the most frequent cause. By means of the compression, air is squeezed out of the vesicle, and the site at which the pressure takes place becomes collapsed.

Great enlargement of the heart may produce atelectasis. This may also occur from large pericardial effusion. A similar condition may arise from an aneurysm of the thoracic aorta.

Atelectasis of the bases of the lung may be caused by upward pressure of the diaphragm, as is the case in abdominal tumors of various kinds. Deformity of the chest due to scoli-

osis of a high grade gives rise to atelectasis corresponding to the convexity of the curvature.

Symptoms.—The respiratory acts are labored and increased, being thoracic in type. There is an inspiratory contraction of the bases of the chest. Upon percussion, a flat note is produced; and upon auscultation bronchovesicular breathing, with moist rales. Often crepitant rales may occur in the vesicles which have collapsed. If the amount of the consolidated area be great, bronchial breathing may be noted.

Prognosis.—This depends upon the ability to remove the cause. In the majority of instances it is unfavorable.

Treatment.—The primary disease should be treated. The patient should be encouraged to take deep inspirations, and change of posture should be frequently and systematically practiced. Cold effusions of the chest are sometimes useful. Strychnin and inhalations of oxygen are beneficial.

PULMONARY HEMORRHAGE.

Two important varieties occur: First, bronchopulmonary hemorrhage, where the blood escapes into the bronchus and is expectorated; and secondly, the condition known as pulmonary apoplexy, in which the bleeding takes place into the lung tissue and in the air-cells. This condition is important from a pathologic and clinical standpoint.

Bronchopulmonary Hemorrhage; Hemoptysis.—This condition is not necessarily a serious one, as it arises under many circumstances and occasionally in young persons apparently in good health. It may consist in bringing up a mouthful of blood, which may be followed by similar attacks for two or three days, then cease without ill effects. This is, however, rare.

In the majority of cases, hemoptysis is an early sign of pulmonary tuberculosis, occurring before physical signs appear—at least before they are well marked. Early in tuberculosis a bronchopneumonia appears, with inflammation of the terminal structures. If it occurs late in the course of pulmonary tuberculosis, it is often due to erosion of an arterial twig or a minute aneurysmal dilatation of a small artery in a cavity. It is apt to be copious, and may be fatal.

Bronchopulmonary hemorrhage takes place in many acute and chronic diseases of the lung, occasionally in the initial stages of croupous pneumonia, where it must not be confused

with the rusty sputum. It results from cancer of the lungs, in abscesses, in bronchiectasis, ulcerative lesions of bronchi, and rarely in acute bronchitis, in plethoric individuals, and frequently in diseases of the mitral valve. It may be the result of an aortic aneurysm. Malignant diseases and parasites are also causes.

There is a form of hemoptysis described as vicarious hemorrhage, occurring in women, in which the hemorrhage is supposed to take the place of the menstrual flow. It is probably an early sign of tubercular involvement, and not due to a vicarious menstruation.

Pulmonary hemorrhage may occur in gouty persons, and it also occurs in the hemorrhagic diathesis, especially in various forms of purpura. Trauma to the chest-wall may be a cause.

Symptoms.—As a rule, the attack begins suddenly, the patient experiencing a sensation of warmth and a salty taste in the mouth, followed by the expectoration of a quantity of bright red blood. Coughing is apt to follow, as the larger bronchial tubes and the trachea may contain blood. The quantity of blood expectorated varies from an ounce to several quarts.

There may be bronchopulmonary hemorrhage without the appearance of blood, as when bleeding takes place into a large pulmonary cavity. Blood-spitting should not be mistaken for blood coming from the stomach.

As a rule, during the period of blood-spitting there is no fever, and if the hemorrhage be profuse subnormal temperature may be present. The pulse is feeble and rapid. The skin may be cold and clammy and the expression anxious. Later, when the small clots are coughed up, a reactionary febrile movement, usually ranging from 100° F. to 104° F., occurs. The previous history is important. The attacks are apt to recur for a few days, and after the last spitting of blood the sputum may be blood-stained for a day or so. This is followed by the appearance of small dark blood-clots. These are simply coagula which have formed at the time of blood spitting and undergone softening. It occasionally transpires that portions of the blood may be swallowed and vomited after a day or two.

The chest should not be examined by the means of physical methods, as the thrombus which has formed in the vessel and prevents the hemorrhage may be dislodged, and the bleeding start anew.

Differential Diagnosis.—

Hemoptysis.	*Hematemesis.*
Blood is bright red and frothy; often coughed up.	Blood is dark, clotted, and often mixed with food; is vomited. Stools may be tarry.
Alkaline in reaction.	Acid in reaction.
Containing air-bubbles.	Air-bubbles absent.
Tubercle bacilli may be present in the blood.	No tubercle bacilli in the blood.

Prognosis.—In the early blood-spitting due to tuberculosis recovery from the hemoptysis is the rule. The later hemorrhage in phthisis, such as occurs into the large cavity, is apt to be fatal.

Pulmonary Apoplexy.—Synonym.—Hemorrhagic infarct. This occurs when blood is effused into the tissues of the lung and the air-cells. As a rule, the process is not diffused, although occasionally it may be extensive. The infarct may be single or multiple. Its most common situation is the lower lobe of the right lung. It results from the blocking of a blood-vessel by a thrombus or embolus. It is most often located at the periphery of the lung, and is wedge-shaped, the apex of the wedge being at the point of the blocking of the artery, and the base toward the pleura. Occasionally the infarct is not wedge-shaped, but irregularly oval, occurring in the substance of the lung, looking like a recent blood-clot. The overlying pleura is mostly involved so that a plastic pleurisy results. The infarct is firm, airless, and the surface raised if situated superficially. On section it is bluish or brownish-red in color and somewhat granular, and microscopically the infarct contains leukocytes and many red corpuscles infiltrated into the tissues and fibrin. The infarct may undergo subsequent changes. It may be absorbed (if not too large), or, if infected, abscess or gangrene may develop. A cyst or scar tissue may replace the area. It is sometimes possible to find the obstruction which has caused the blocking.

Emboli may result from right-sided heart lesions or from the breaking up of a thrombus in the peripheral veins, reaching the right side of the heart, and enter the pulmonary artery. Rarely thrombosis of the pulmonary artery, due often to sluggish circulation from valvular disease of the heart and weakened muscle, is a cause of pulmonary apoplexy. It is more common in men than in women. It is more common between the ages of fifteen and thirty-five, and is rare in the extremes of life.

Symptoms.—The clinical phenomena are not definite. There may be a chill, dyspnea, pain in the side, and unconsciousness, and often death. Upon examination, dullness upon percussion and a friction sound may be present.

Treatment of Pulmonary Hemorrhage.—The patient should be put to bed and absolute bodily and mental rest enjoined. No food, and especially no fluids, should be administered. Ice-bags may be placed upon the chest. The hemorrhage ceases by the formation of a thrombus in the affected region, and the formation of this thrombus can best be attained by lowering blood pressure and the administration of opium or its derivatives. If the heart's action is tumultuous, small doses of tincture of aconite may be given. The administration of salt is useless, and only increases the thirst. All measures which are of advantage in internal bleeding are of use in this condition. Systematic purging is often good practice.

BRONCHOPNEUMONIA.

Synonyms.—Lobular pneumonia; catarrhal pneumonia; capillary bronchitis.

Definition.—This disease is in the majority of instances a secondary affection, following bronchitis. The inflammatory process, from continuity of structure, proceeds from the large bronchial tubes into the finer ones, and finally affects the vesicular structure of the lungs.

Etiology.—It occurs most frequently in the extremes of life, infants and the aged being most subject to the affection. According to Aufrecht, scrofula and rickets predispose. The infectious diseases, especially those that are accompanied by bronchitis, such as measles, pertussis, influenza, diphtheria, enteric fever, etc., are likely to produce the disease, especially when the vital forces are greatly lowered. Most cases develop in the winter and spring months.

In many severe acute and even in chronic diseases the malady is apt to occur. In some instances, foreign material, sputum, and particles of food are likely to find their way into the trachea, and set up inflammatory changes which rapidly infect the finer bronchi and alveoli, thus producing bronchopneumonia. This condition is called inhalation, insufflation or deglutition pneumonia. It is likely to happen in the low fevers and in diseases characterized by the so-called "typhoid state."

Micro-organisms of various kinds, pneumococcus, strepto-

coccus, staphylococcus, Klebs-Löffler bacillus, bacillus of influenza, bacillus of tuberculosis, and others, may find their way into the lobules and give rise to the affection.

Pathology.—The pathologic changes are chiefly those encountered in catarrhal inflammations. Consolidated masses of various sizes and shapes are found in different parts of the lungs. This frequently happens in pulmonary tuberculosis, in which case the tubercle bacillus is the excitant.

The lungs are seen to be distended and injected, and do not collapse readily. Rounded areas of red patches, slightly elevated, may be seen beneath the pleura. The pleura is often opaque and covered with a delicate fibrinous material, but this is absent in many cases. Masses of various sizes may be felt in the lung substance. On section of the lung the nodules are flattened, or of a reddish-gray or dark red color. Upon pressure a small amount of yellow viscid fluid escapes. The bronchi are filled with an inflammatory exudate, or sometimes with the foreign material which has caused the condition.

Surrounding the nodules the lung tissue is often emphysematous, and here or there may be seen collapsed areas (atelectasis). Both lungs are usually involved in the process, and any part of the lung is liable to be affected.

The smaller and medium-sized bronchi are the seat of a catarrhal inflammation and filled with a mucopurulent exudate which may be squeezed out. The lung crepitates in most parts, and floats when placed in water; the consolidated areas, when excised and thrown in water, sink.

Microscopically, the terminal bronchi and alveoli show varying quantities of mucous exudate mixed with epithelial cells, leukocytes, few red blood-cells, and little if any fibrin. The blood-vessels are dilated, and the lung structure is infiltrated with leukocytes.

Symptoms.—Primary Form.—In rare instances bronchopneumonia begins as an acute primary affection with the symptoms of a severe bronchitis. The patient has malaise, cough, dyspnea, and pain. Fever may range between 102° and 103° F. The expectoration is not characteristic, but often the ordinary mucopurulent variety, never mixed with blood, or rusty. The physical examination shows rales, bronchovesicular breathing, and small areas of consolidation in both lungs, surrounded by tympanitic areas. The affection may last two or three weeks, the disease ending by lysis, and never by crisis.

Secondary Form.—The onset of the disease is gradual, the early symptoms being those of the preceding bronchitis. Symptoms which call attention to the fact that bronchopneumonia has developed are a rise in the temperature to 104° F. or 105° F., increased dyspnea, rapid, feeble, running pulse, cough becoming more difficult, development of cyanosis, and a change in the physical signs.

The respiration, especially in children, is from 60 to 80 per minute. This is accompanied by painful cough. In very young children there is no expectoration, and even where the sputum is present it is not characteristic. The patient is restless, apathetic, and frequently slightly somnolent.

The pulse is extremely rapid, reaching a frequency of from 140 to 160 per minute. Fever is always present, and usually high, from 103° F. to 105° F., but the temperature curve is not typical.

Physical Signs.—Inspection.—Upon inspection, increased respiratory effort will be noticed upon both sides of the chest, also some slight cyanosis.

Palpation.—This may develop limited areas of increased vocal fremitus.

Percussion.—Upon percussion, small areas of dullness, surrounded by limited tympanitic area in both lungs, especially posteriorly, will be noted.

Auscultation.—The most typical signs are present upon auscultation. Bronchovesicular breathing and fine moist rales, most often subcrepitant in character, are diffused over the chest. To these will be joined the rales of the accompanying bronchitis of the larger tubes.

Complications and Sequels.—The course of the disease is slow. In cases that terminate favorably it lasts from three to four weeks, often longer, during which time remissions and exacerbations are apt to occur.

The disease may terminate in tuberculosis; especially is this apt to occur when the bronchopneumonia takes place during the course of pertussis or measles.

Occasionally abscesses and gangrene of the lung follows. Should the pleura become involved, signs of the pleurisy, with or without effusion, and occasionally even empyema may develop. It may sometimes be followed by chronic interstitial pneumonia.

Diagnosis.—As a rule the diagnosis is not difficult. The persisting bronchitis, with a sudden rise in temperature, in-

creased respirations, and the physical signs, usually determine the diagnosis. The disease must, however, be differentiated from croupous pneumonia, which it may resemble in some degree.

Differential Diagnosis.—

Bronchopneumonia.	*Croupous Pneumonia.*
The disease is secondary following bronchitis, and begins gradually.	A primary affection, beginning suddenly, often with marked chill.
A bilateral disease.	Most often unilateral.
Temperature not typical.	Typical temperature.
Prolonged disease ending by lysis.	Short affection, ending by crisis.
Affecting lobules in both lungs.	Affecting one or more lobes of the lung.
Sputum not characteristic; no herpes.	Sputum rusty; herpes common.
Subcrepitant rales; bronchovesicular breathing.	In first stage, crepitant rales; second stage, bronchial breathing; third stage, crepitus redux.

Prognosis.—Bronchopneumonia is always a serious affection. In children under two years, 50 per cent. of the cases are fatal; under five years, one-fourth of the number of children affected die. It is equally serious in the aged. The disease is extremely rare during the middle periods of life.

Treatment.—The prophylaxis is most important. A child affected with acute bronchitis should be carefully guarded, and the possibility kept in mind that the case may develop into bronchopneumonia. Proper attention should be given to slight nasal catarrh and mild coughs.

In the treatment of bronchopneumonia itself, when the temperature becomes high, cyanosis shows itself, and somnolence is threatened, a warm bath with cold effusions to the head is useful. When the pulse becomes weak, alcohol is indicated. The application of ice poultices, sinapisms, and turpentine stupes to the chest is of benefit. Bleeding is not indicated. Inhalations of steam that has been medicated by compound tincture of benzoin or camphorated tincture of opium are useful. In strong children in whom there is great secretion in the bronchial tube, which is with difficulty brought up, emetics should be used from time to time, but these should not be continued throughout the course of the disease. Narcotics should not be administered to very young children. Stimulating expectorants, such as the salts of ammonia, are often useful. Minute doses of strychnin and inhalations of oxygen are of benefit. A mild purge at the onset frequently gives great relief. The diet should be a light, nutritious one.

CHRONIC FIBROID PNEUMONIA.

Synonym.—Cirrhosis of the lung.

Definition.—This occurs under a variety of circumstances in which there is an extensive formation of fibrous tissue in the lung, extending from the bronchi and blood-vessels, the pleural covering, and interlobular tissue. It is usually the result of a secondary process, and may be either localized or diffused.

Etiology.—A localized formation of cicatricial tissue replaces destruction of lung which may be occasioned by abscess, gangrene, injury, etc.

The encroachment by parasites which have encysted themselves, abscesses, tubercles, gummata, aneurysms, and tumors, are often prevented from spreading or checked by fibrous tissue in their immediate vicinity. This condition is then more often beneficial than injurious. It often occurs during the course of chronic bronchitis, the growth of fibrous tissue extending into the lung from the bronchial walls. Dilatation of the bronchi, obliteration of the bronchioles, and emphysema are often associated.

Tuberculosis, syphilis, croupous and bronchopneumonia, and the inhalation of fine particles of coal dust, limestone, chalk, silica, and iron may give rise to cirrhosis of the lung.

Pathology.—It is most frequently a unilateral disease, sometimes bilateral, and often localized to small areas. The involved lung is much contracted, dense, heavy, and very tough, and offers great resistance to the knife. The surface of the lung is irregularly nodular.

The pleura is usually greatly thickened, revealing the same general pathologic process. The adjacent organs, heart, opposite lung, and liver, are displaced.

Upon section the cut surface presents a smooth appearance, and the color is grayish-red ; a yellow fluid exudes. The lung may be pigmented, depending upon the character of the irritant. Microscopically, a great increase in fibrous connective tissue will be noticed, in some instances so profuse that the alveolar structure has entirely disappeared. The new-formed connective tissue is in some instances marked about the bronchi, in others around the blood-vessels, or may extend from the pleura. From the narrowing and destruction of numerous blood-vessels, during the course of their change, the flow

of the blood through the lung is impeded, giving rise to hypertrophy and dilatation of the right heart.

Compensatory emphysema of the unaffected lung is a natural sequel. Pericarditis may occur during the course of the process.

Symptoms.—The disease frequently begins as an ordinary acute pneumonia, terminating either by crisis or lysis. Occasionally, a subfebrile temperature may remain for several weeks. Dyspnea and cough continue, and the sputum becomes mucopurulent. If the case does not terminate fatally, the fever subsides, the cough lessens, and the patient improves, but a retraction of the affected side of the chest takes place, which remains permanently. Cough, dyspnea, cyanosis, and edema are likely to follow from the affection of the right heart. A similar condition has been described as the result of congenital syphilis, involving both lungs.

Physical Signs.—Inspection.—The heart is displaced and drawn toward the affected side. Compensatory emphysema is usually noted upon the healthy side. The diseased side of the chest is retracted; the intercostal spaces are almost obliterated by the ribs coming closer together.

Palpation.—Respiratory movement is restricted upon the affected side. The vocal fremitus is usually increased except when the pleura is greatly thickened, when it may be absent altogether.

Percussion.—Great differences of the note are elicited upon percussion. Most often there is dullness and even flatness, but a tympanitic note may be elicited if there is a dilated bronchus in the retracted area.

Auscultation.—The breath-sounds are distant and feeble. Bronchovesicular and even distant bronchial breathing may occur. All kinds of moist and dry rales are heard, often accompanied by some friction sounds.

Diagnosis.—Early the disease can not be differentiated from the delayed resolution occurring from croupous pneumonia. Only when retraction of the chest takes place can the condition be diagnosticated.

Prognosis.—The prognosis as to life is favorable, but the condition is incurable. The patient is very liable to recurring attacks of bronchitis, and bronchiectasis is very apt to arise. Death often results from failure of the right heart.

Treatment.—A nutritious diet is important. The patient should have the best and most substantial food. The resi-

dence in a high and dry altitude is of great use, otherwise the treatment is symptomatic. Tonics are often useful.

CONGESTION OF THE LUNGS.

There are two varieties of this condition : Active and passive.

Active Congestion.

Hyperemia or active congestion occurs from increased action of the heart, inhalation of irritating chemic substances, heated air, dust, etc. Obstruction to the blood-current in one lung often causes more blood to be driven into the other lung, giving rise to active congestion. Hyperemia occurs in pneumonia, pleurisy, pressure from tumors, and like conditions.

It may arise from exposure to extreme heat or cold, especially after exertion, such as public speaking under great excitement, undergoing violent exertion, passing from a heated and close auditorium into the cold air.

The involved area is dark red in color, heavier than normal, but floats when placed in water. Upon section blood drips from the cut surface. Some of the alveoli may contain blood.

Symptoms.—These are by no means well defined. There may be chill followed by fever, the temperature rising to 101° F. or 103° F., with cough, dyspnea, and pain in the side. The physical signs may show enfeebled respirations, impairment of resonance, and fine moist rales. It frequently terminates fatally.

Passive Congestion.

Passive congestion is subdivided into (a) mechanical, (b) hypostatic.

Mechanical passive congestion occurs where there is an obstacle to the return of the blood, as in valvular disease and myocarditis, the disturbance of blood-pressure resulting in the accumulation of blood in the lungs. Tumors of the mediastinal tissues may interfere with the circulation. When the congestion has persisted for some time, brown induration of the lung results.

If due to cardiac disease, no symptoms appear so long as compensation is maintained, but with rupture of compensation, symptoms of cough, dyspnea, expectoration, and sometimes hemoptysis appear.

Hypostatic congestion occurs in adynamic conditions, especially in the low fevers. The bases of the lungs become deeply congested, partly from the action of gravity and partly due to the enfeebled circulation, causing the blood to accumulate in the bases posteriorly. The lung is dark red in color and engorged with blood; some portions of it may be atelectatic, and may sink when thrown into water. This condition has been termed "hypostatic pneumonia."

Treatment.—The treatment consists in removing the underlying causes if possible. In active congestion free bleeding is often of great value.

PULMONARY EDEMA.

Transudation of the watery elements of the blood through the walls of the blood-vessels into the alveoli, often the bronchi and interstitial tissues of the lungs, gives rise to what is termed "pulmonary edema."

The edema may be general or local.

General Edema.—All causes which give rise to active and passive congestion may produce edema. It is frequently the terminal event in many chronic affections, especially cardiac, renal, pulmonary, and cerebral diseases, also from the cachexias and anemias.

Local edema may result from local disturbance of the blood supply, as infarcts, tumors, etc., also from inflammatory conditions.

Pathology.—The edematous lung is heavier than the normal, but floats when placed in water. Crepitation is elicited. The color of the lung varies, depending upon the causation; if resulting from renal disease, it is often of the normal color; when from congestion, it is dark red. Upon section serum escapes from the cut surface; this may be frothy or bloody. (The lung is spoken of as being "water-logged.") The edema is most marked in the dependent portions. The general factors concerned in the development of edema are disturbance in the blood pressure, change of the blood composition, and changes in the vessel walls.

Symptoms.—As the condition is usually secondary, an aggravation of the existing symptoms of the disease takes place. The dyspnea increases; the cough becomes more urgent and troublesome; large numbers of moist rales of all sizes are heard, particularly at the bases of the lungs; and

the tracheal rale, known as the "death-rattle," is in evidence in those terminating fatally.

Treatment.—The treatment is that of the primary disease. There should be active purging, especially if cyanosis be absent. Hypodermics of atropia in large doses frequently repeated have been found of use in some cases.

GANGRENE OF THE LUNG.

All conditions which lead to abscesses may lead to gangrene, where necrotic areas are followed by putrefaction. It occurs in diabetes mellitus, pneumonia, from new growths, infarcts, and from septic emboli. The condition is found more commonly in the lower lobe. It appears in debilitated subjects, and frequently after aspiration pneumonia.

Pathology.—The appearance of gangrene is characteristic. The lung is softened and diffluent, and of a dirty grayish-black color. The odor of the lung is very foul and of a penetrating character. An inflammatory process is always found around the area of gangrene.

Symptoms.—Cough accompanied by expectoration, which is abundant, thin, and foul-smelling, should call attention to gangrene of the lung. Fever is always present, the patient lying upon the affected side. There is often hemoptysis. The sputum, upon standing, collects into three layers—a top layer, which consists of mucopurulent material; a middle layer, which is thin and watery; and a lower layer, which consists of pus with greenish threads in it. Microscopically, leukocytes, shreds of lung tissue, especially elastic fibers, fat crystals, and bacteria are present.

Upon percussion over the affected area dullness is usually present. Upon auscultation there is bronchial breathing, and if excavation occur, signs of cavity will be noted.

Prognosis.—The prognosis is very unfavorable.

Treatment.—The treatment is expectant symptomatic.

ABSCESS OF THE LUNG.

Abscesses may result during the course of pyemia, and may follow broncho- and croupous pneumonia; they may result by extension, as from abscess of the liver, subphrenic abscess, and empyema. Abscess also frequently accompanies chronic tuberculosis. Trauma and the introduction of foreign bodies

may sometimes give rise to the conditions. Carcinoma in which ulceration exists may give rise to marked suppuration of the lung.

The abscesses vary greatly as to size and distribution.

The Friedländer bacillus, diplococcus of pneumonia, staphylococcus, bacillus of influenza, and streptococcus are the most common micro-organisms that have been found in the pus.

Symptoms.—These are not characteristic. The history must be taken into account. Fever of the septic type is present. The physical signs of a cavity are usually noted. Often there is expectoration of pus, which is foul-smelling, and under the microscope contains elastic fibers. Leukocytosis is present in nearly all such cases. If the condition affect the pleura, the signs of pleuritis will be observed.

Prognosis.—Is always guarded. In simple abscesses it may be favorable; in embolic abscesses it is always grave.

Treatment.—The treatment should be supportive. If the abscess is accessible, surgical interference is necessary.

PNEUMONOKONIOSIS.

Definition.—A disease of the lungs due to the inhalation of particles of dust, often followed by fibroid changes.

Etiology.—Occupations which expose the individual to the inhalation of dust particles, such as coal-mining, stone-cutting, knife-grinding, and the work of millers predispose to the disease.

Pathology.—In the inhalation of dust particles, irritation is first set up in the epithelial lining of the bronchi, producing a catarrhal inflammation which is usually chronic. Later the dust particles are deposited in the lymphatic spaces of the lung, a proliferation of the connective-tissue cells results, and finally fibrous tissue forms, giving rise to interstitial pneumonia.

In this new-formed tissue the particles are held. In the case of coal dust, the lung is black; if due to chalk or limestone it is of a light-gray color.

The lung usually shows diffuse involvement, but the infiltration may be more localized in some instances. The bronchial lymphatic glands almost invariably show infiltration. The lungs are usually increased in weight, especially so in anthracosis. Crepitation is commonly present throughout. In some

instances marked interstitial changes are present, the alveolar and bronchial tissues being replaced by the new formation.

Infection may occur under these conditions by various micro-organisms, particularly by the tubercle bacillus. Abscess sometimes results. The pleura are usually thickened. Emphysema may follow, and bronchitis is generally present.

Varieties.—(1) **Anthracosis.**—This condition is due to the inhalation of particles of coal, and is sometimes called "miners' phthisis." (2) **Siderosis.**—This condition is due to the inhalation of particles of metal, especially fine steel. It is present in knife-grinders or in those exposed to the inhalation of small fragments of steel and of iron. It has been called "knife-grinders' phthisis," and the average duration of life in persons affected with this form of pneumonokoniosis is about six years. (3) **Chalicosis.**—This is due to the inhalation of small mineral particles, and occurs in occupations involving the chipping of minerals, such as millstones, etc. (4) **Millers' Phthisis.**—This is found in occupations in which cereals are ground.

Symptoms.—The symptoms come on very gradually after many months or even years, first characterized by a more or less well-defined bronchitis. This may be followed by the development of an interstitial pneumonia, or the occurrence of tuberculosis. The sputum often contains the particles inhaled, as well as such characteristics encountered in the sputum of chronic bronchitis.

Diagnosis.—The diagnosis depends upon the occupation of the patient, the gradually failing health, the condition of the sputum, and sometimes signs of consolidation, especially at the apex.

Prognosis.—The prognosis depends upon the ability of the patient to change his occupation. If the disease be not too far advanced, cure may result.

Treatment.—Prophylaxis consists in means devised to arrest the inhalation of dust particles. The treatment of the disease rests upon general principles.

SYPHILIS OF THE LUNGS.

Definition.—Syphilis of the lungs occurs in two forms: (1) *The congenital form*; (2) *the acquired form*. The acquired form is associated with the formation of gummata, and sometimes sclerotic changes.

1. **Congenital Syphilis.**—This shows itself in the form to which the name *pneumonia alba*, or white pneumonia, has been given. On section the lung is light-gray, nearly white in color, and almost completely airless.

Microscopically there is wide-spread round-cell and spindle-cell infiltration, and fully developed connective tissue in the interalveolar and interlobular substance, with more or less compression of the bronchioles and alveoli. Some of the cells contain numerous fat drops, the structure having undergone fatty degeneration. When pneumonia alba is present, the child is either born dead or dies shortly after birth. The symptoms are indefinite, although consolidation may be revealed upon examination. Evidences of syphilis in other parts of the body will be found. Clinically, white pneumonia can not be differentiated from bronchopneumonia.

2. **Acquired Syphilis.**

Pathology.—Changes in the lung are due to the formation of gummata, usually accompanied by diffuse interstitial lesions. The gummata are always characteristic, but the condition is extremely rare. They occur in the form of firm nodules, varying in size from a pea to a large apple, with a pale yellow cheesy center of firm consistency, surrounded by a translucent, grayish tissue, ending in an area of injected lung substance. The gummata are likely to appear near the root of the lung, often connected with the bronchi; in this way symptoms of pressure occur. If there are no symptoms of pressure, the condition can not be diagnosticated. Secondary changes which may be of syphilitic origin are interstitial changes, due to the growth of dense connective tissue. The connective tissue may also form in the bronchial wall and peribronchial tissue, producing nodular masses. In this connection bronchopneumonia may result. Sclerotic changes of the blood-vessel walls are also present.

Symptoms.—In symptoms referable to the respiratory organs, in the person suffering from acquired syphilis, when other diseased conditions can be eliminated, syphilis of the lung should be suspected. The most common symptom is dyspnea, increased by exertion and becoming worse as the disease progresses. Cough is present, usually with mucopurulent expectoration.

Physical Signs.—The physical signs are those of stenosis of the bronchial tube, or bronchiectasis, or similar signs due

to profound bronchitis or bronchopneumonia. The physical signs, as a rule, are obscure.

Prognosis.—The prognosis is unfavorable.

Treatment.—The treatment is that of syphilis in general—mercury and iodid of potassium.

NEW GROWTHS OF THE LUNG.

New growths of the lung are rarely primary; they are most often secondary, the evidences of primary growth being found elsewhere in the body, and being transferred by means of the blood or lymphatics.

Primary Tumors.—Of the benign connective-tissue tumors, fibromata, lipomata, and chondromata have been observed, but they are rare. Of the epithelial benign tumors, adenomata have been recorded. Of the embryonic connective-tissue tumors, various sarcomata have been found, but are usually secondary.

Carcinomata as primary growths have also been found; these are usually massive growths situated at the base of the lung. They show a tendency to ulceration and degeneration (as is common in carcinoma). They may be of the cylindric variety, or if they spring from the squamous epithelium of the alveoli, are of that type. The neighboring lymphatic glands are involved.

Secondary Tumors.—Secondary tumors are more common in sarcoma than in carcinoma.

Symptoms.—The symptoms are those of pressure, the patient complaining of dyspnea, cough, and expectoration; and occasionally hemoptysis occurs. There may be difficulty in swallowing, due to pressure upon the esophagus, or the mass may press upon the recurrent laryngeal nerve, causing paralysis of the vocal cords.

When there is sharp stitch-like pain, the pleura is involved. *Physical examination* may reveal bulging of the affected side, due to the growth.

Percussion gives flatness, and *auscultation* shows absence of breath-sounds. Enlargement of the glands in the axilla in case of carcinoma and dilatation of the veins of the neck are of value in diagnosis.

Prognosis.—The duration of the disease is from one-half a year to a year and a half in cases of the malignant tumors.

Treatment.—The treatment is symptomatic.

PARASITES.

The echinococcus or hydatid cyst occasionally appears in the lung. It usually represents the extension from the liver, the cyst having ruptured through the diaphragm. Cough and hemoptysis are sometimes present; however, the signs and symptoms are not characteristic.

DISEASES OF THE PLEURA.

PLEURISY.

Definition.—Pleurisy is an inflammation of the whole or a part of the pleura. The condition may be either *acute* or *chronic*.

The disease may be classified into *dry* or *adhesive*, and *pleurisy with effusion*. Pathologically the disease may be classified into *fibrinous*, *serofibrinous*, *purulent* inflammation, and *fibrous or chronic pleurisy*.

The disease may be *primary* or *secondary*. The clinical classification is divided into the acute and the chronic forms.

DRY, FIBRINOUS OR PLASTIC PLEURISY.

Etiology.—The condition is often the result of prolonged exposure to cold. It may often occur in the rheumatic diathesis, in many of the infectious diseases, and in rare instances may be due to syphilis. Traumatism is also a prominent cause.

In the majority of instances the disease is secondary, due to some affection of the lung, one of the principal causes being pulmonary tuberculosis; next in frequency, croupous pneumonia and infarcts of the lung. It occasionally occurs in connection with pericarditis.

Pathology.—The pleura is usually involved to a limited extent, although the entire area may be affected. The earliest change noted is a reddening of the surface. The pleura loses its luster, becomes rough and dry, and later the fibrinous exudate will be formed upon the surface, which varies greatly as to extent and thickness. The visceral and parietal layers may become temporarily adherent as a result. The earliest change is hyperemia, followed by the migration of leukocytes

and the pouring out of liquor sanguinis, which finds its way to the free surface, where fibrin is formed. This results from the action of some ferment upon the fibrinogen of the blood. There is also a proliferation of the fixed connective-tissue cells of the part, and red blood-cells may be found in the perivascular tissues. The endothelial cells show granular degeneration early in the stage of inflammation, and reveal desquamation.

Fibrinous pleurisy frequently terminates in pleurisy with adhesions. When the exudate is well formed, both surfaces of the pleura may be adherent, and in this framework of fibrin new blood-vessels appear, springing from the visceral and parietal layers. Connective-tissue cells proliferate in this area; they elongate, forming fibroblasts, and finally fully formed connective tissue is developed, the fibrin being absorbed in the greatest number of instances. This results in the formation of permanent adhesions. Whether the exudate is ever completely absorbed, leaving the serous membrane in a healthy condition, is doubtful.

Symptoms.—The disease may either come on acutely or insidiously. The acute form begins with sharp, shooting pain upon the affected side. Occasionally, however, the pain is felt in the abdomen, and even upon the opposite side. A chill rarely occurs. There is slight fever, the temperature ranging from 100° F. to 102° F. This fever soon declines, often reaching the normal upon the second day and remaining so throughout the course of the affection.

The respirations are slightly increased in number, and there may be a dry cough, which the patient endeavors to suppress. The pain itself rarely lasts longer than one or two days, giving place to a feeling of soreness upon the affected side. The majority of such cases terminate in speedy recovery.

The disease may have a tendency to recur, or if it begins gradually, great pleural thickening develops without pain. The greater number of such cases are tubercular in origin. The symptoms in this form are mild, although this is not invariably the case.

Physical Signs.—Upon inspection the respiratory excurses upon the affected side may be decreased. Occasionally upon palpation the friction fremitus is felt. There is no change from the normal on percussion. The important sign is the friction sound which is heard upon auscultation. It is described as a grazing, rubbing or rasping sound, sometimes resembling

the creaking of new leather. It is heard either with inspiration and expiration or both, and is not affected by cough. It may be increased by pressure of the stethoscope. The friction sound bears no relation to the presence of pain. There may be wide areas of friction with little or no pain.

Pleuropericardial Friction.—If the pleurisy occur upon the left side, a friction sound may be heard which is synchronous with the beat of the heart. This is due to the rubbing of the pericardial sac against the roughened pleura, and can be distinguished from ordinary pericardial friction by being increased at the height of inspiration and by its limitation to the left border of the pericardial area.

Diagnosis.—The diagnosis depends upon the sudden onset with pain, slight fever, and presence of a friction sound upon auscultation.

Differential Diagnosis.—

Plastic Pleurisy.	*Intercostal Neuralgia.*
Pain somewhat diffused, not limited to a certain area.	Pain particularly localized to the exit of the intercostal nerves.
Slight fever and cough.	No fever and no cough.
Herpes absent.	Herpes in the affected area common.
Friction sounds present.	No friction sounds.

SEROFIBRINOUS PLEURISY.

Synonym.—Pleurisy with effusion.

Etiology.—In the present state of knowledge, it is safe to say that the majority of primary pleural effusions are tubercular in origin. Cold and exposure, especially exposure to a chilly, moist atmosphere, may be considered as the predisposing causes, although some authorities entirely deny the etiologic influence of cold.

Other conditions which cause pleural effusion are croupous pneumonia, the pneumococcus being found in serofibrinous effusions. Acute rheumatic fever is sometimes accompanied by pleural effusion. It may occur in the course of syphilis. In enteric fever pleural effusions take place, and in the infectious diseases of childhood the condition is not uncommon, although in the greater number of these cases the effusion is likely to be purulent in character. Traumatism must also be given a place in the etiology. Secondary pleural effusions sometimes occur as the terminal event in chronic diseases of the heart and kidney.

In aneurysms, new growths, abscesses, and hydatids of the liver, peritonitis, and in ovarian cysts, serofibrinous pleurisy

may develop. The condition is most common between the twentieth and the fortieth years, but it also occurs in infancy. It is perhaps somewhat more frequent in men than in women. The disease is more prevalent in the cold season.

Pathology.—Serofibrinous pleurisy may be the further development of the dry stage, although in many instances it begins at once as a serous exudation. The liquid exudate is heavier than the fluids found in transudates, and it contains flakes or shreds of fibrin. The specific gravity of the exudate is usually above 1018, in transudates being below this.

The character of the exudate is pale yellow, sometimes brownish yellow, transparent, and is occasionally a slightly opaque, odorless fluid. A quantity of fibrin is usually suspended in it, which may coagulate spontaneously several hours after its withdrawal. It is alkaline in reaction. The occurrence of red corpuscles is most commonly encountered in exudates due to tuberculosis and new growths of the pleura.

Microscopically, white and red corpuscles and detached endothelial cells are found. Sometimes the erythrocytes are found in great numbers; it is then called a hemorrhagic exudate. The amount of fluid contained in the pleura may vary from a small quantity to several liters.

The pleura itself may show an extensive fibrinous exudate. The lung is pressed backward and upward, and may be atelectatic. Adjacent organs—the heart, spleen, stomach, and liver—are displaced, depending upon the amount and position of the fluid, whether it be upon the right or the left side.

Symptoms.—Prodromes may occur, such as pain in the side, and general malaise, or the condition may begin abruptly with chill, fever, and severe pain from the onset. Sometimes, especially in children, it happens that the patient complains of symptoms of depression, weakness, loss of appetite, palpitation and breathlessness upon exertion, which is mistaken for prolonged enteric fever. Upon physical examination, however, an extensive pleurisy is found.

The pain does not always occur, but when present it is apt to be distressing, and is referred to the region of the nipple, or axillary region. It is aggravated by cough, by deep breathing, and by firm pressure upon the chest. Dyspnea is common, especially with large pleural effusions. This is due to encroachment upon the respiratory surface of the lung. If the effusion develops slowly, the lung may be entirely compressed without dyspnea occurring, except, perhaps, upon

exertion. In large effusions the patient lies upon the affected side, or upon the back so as not to encroach upon the lung or the unaffected side. Fever may or may not be a symptom; when present, it is slight, the temperature ranging from 100° F. to 102° F. The pulse is slightly accelerated.

Physical Signs.—Inspection.—Some degree of immobility of the affected side is present; this may vary in proportion to the amount of fluid in the pleura. In massive effusions the affected side of the chest becomes comparatively immobile, and the unaffected side shows an increase in the respiratory excursis, taking upon itself more work. There may be some increase in volume of the diseased side; this is especially true in large effusions with obliteration of the intercostal spaces, which rise up to the level of the ribs, showing a contrast in the inspirations between the affected and the unaffected side.

In moderate or large right-sided effusions, the apex of the heart is displaced to the left and upward, found occasionally even in the fifth intercostal space to the left of the mammillary line. The impulse of the heart may be found to the left as far as the anterior axillary line. In left-sided effusion the impulse may be entirely absent, due to the fact that the apex of the heart is behind the sternum, or it may be visible in the third or fourth intercostal space of the right side.

Palpation.—Palpation confirms the signs obtained upon inspection as to the restricted movements upon the affected side. It shows that the intercostal spaces bulge, and determines the position of the cardiac impulse. In serofibrinous pleurisy, edema of the chest is rarely present, this being determined by palpation. In empyema this important physical sign is sometimes present. Any evidence of fluctuation, such as occurs in ascites from effusion into the peritoneal cavity, is an exceedingly rare occurrence in serofibrinous pleurisy. The tactile or vocal fremitus over the side of the effusion is enfeebled or abolished; in young children, on the contrary, it may be retained, as also in individuals with thin chest-walls. The explanation has been given that the vocal fremitus may be transferred through very thin walls over the unaffected side laterally, the vibrations being carried over to the opposite side. In loculated or pocketed serofibrinous effusion there are lines of tissue running from the peripheral pleura to the costal pleura, old adhesions having taken place (the lung being separated by the effusion). These lines of tissue may transfer the vibrations to the surface of the chest, producing

vocal fremitus upon the affected side. A bronchus may be pressed upon by a rib, and the fremitus be carried along the line of the rib, or in some instances the lung may be held to certain areas of the chest from old pleural adhesions, giving rise to localized fremitus. These signs are often very confusing. *Mensuration* is a sign of very little value.

Percussion.—Over the pleural effusion the percussion note is flat, and extending toward the apex, becomes clear and tympanitic in character, known as "Skodaic resonance." The larger the effusion the greater the amount of flatness. In large pleural effusions, posteriorly a curve of an S shape may be made out. According to Ellis, "this curve begins with medium effusions relatively low down in the back, passes outward from the vertebral column, and soon turns upward, and proceeds obliquely across the back to the axillary region, where it reaches its highest point. Thence it advances in a straight line, but with a slight descent to the sternum." As the effusion increases in amount, the curve rises and flattens so that the S-curve disappears, when the effusion reaches the second rib. The curve is modified by pleural adhesions and pathologic changes of the lung, such as consolidation and emphysema. The most characteristic signs of a moderate and large effusion are displacements of organs, particularly of the heart and mediastinum. This may be made out by palpation and percussion.

Reference to the changes in the heart in right- and left-sided effusions has already been made. Depression of the diaphragm also occurs. It may be referred to the right side from the position of the lower margin of the liver, which may in extreme cases be as low as the level of the umbilicus. On the left a moderate degree of depression can easily be detected. Obliteration of "Traube's semilunar space" occurs. (See p. 96.) Any marked depression of the diaphragm by the fluid will cause a perceptible diminution in the width of this zone. In extreme cases, not only may the splenic dullness be entirely obliterated, but the diaphragm may sag below the ribs as a prominent tumor. As a rule, these characteristic signs of depression of the diaphragm are only pronounced in very large pleural effusions.

Upon **auscultation,** vocal resonance is absent over the flat area. The respiratory murmur is decreased or absent over the fluid. When the fluid diminishes, the voice sounds may be heard, but are distant and diffused. Egophony is rare.

ATYPICAL FORMS.

Resorption, or the artificial removal of a portion of the fluid, is indicated by a drop in the line of flatness, and by the diminution of intercostal tension and the rise in the diaphragm. Respiratory sounds and fremitus show various modifications tending toward a return to normal conditions. *All these changes are, however, much less marked than might be expected, and especially is the return of the heart to its normal position slow and retarded.* As the effusion disappears the friction sounds return, sometimes with even more intensity than at the beginning. Large numbers of crackling rales are heard, which, according to Traube, are due to the forcible entrance of air into the group of air-vesicles, which are opened as absorption progresses.

Fibroid Thickening.—Fibroid thickening with retraction of the chest has already been mentioned.

Encapsulated Effusions.—These do not produce displacements, as a rule, the other signs being the same as in ordinary effusions, except that they are less pronounced. Fremitus may be present for reasons already described.

The mobility of pleural effusion on change of posture is still under discussion. Very large effusions are immovable; moderate effusions reaching to the third or fourth rib in front, if they change their position at all, move so slightly as to be scarcely perceptible, and if such alterations do occur, require time, and can not be diagnosticated at once.

PURULENT PLEURISY; EMPYEMA.

Etiology.—The essential difference between a serofibrinous and a purulent effusion usually consists in the presence in the latter of certain micro-organisms in considerable quantity, and many pus-cells. This has been abundantly proven by the investigation of some of the best bacteriologists. It is probable that most empyemas are caused by two or three particular forms, and when they give rise to the disease the clinical course may vary. It is possible that empyema may occasionally be a primary affection, but in the majority of cases the disease is secondary.

The most frequent organism producing the condition is the streptococcus pyogenes. The sources of the invasion into the pleura are from the lung; thus pleurisy is associated with influenza; ulcerative processes as from tuberculosis, abscesses,

gangrene, infarcts, bronchiectasis, and cancer; affections of the skin, lymph-glands and breast, especially cancer of the breast; also from the mediastinum, such as pericarditis; abdominal suppurations, particularly puerperal metritis; also from abscesses of the liver, stomach, or the bowels; the acute affections, as diphtheria, influenza, scarlet fever, erysipelas, and puerperal fever.

To produce empyema from these diseases, it is necessary that the micro-organism (the most frequent being the streptococcus) should invade the pleura in considerable numbers, and particularly that there should be some focus, close to or communicating with the pleura, where the conditions are favorable for their multiplication. (The injection of small quantities of a pure culture into the pleural cavity has proved innocuous.)

Next in importance in producing empyema is the pneumococcus of Fränkel. This organism has been found in all empyemas due to croupous pneumonia. It is sometimes found in empyema following bronchopneumonia, as also in empyemas which are apparently primary.

Tubercular empyema may arise from tubercular affection of the pleura, being usually chronic in character, but the effusion is more likely to be serofibrinous than purulent.

Other micro-organisms are exceptionally found—the staphylococcus, the bacillus of Eberth, and occasionally the encapsulated bacillus of Friedländer.

Saprophytic bacteria are often the determining cause of the fetid and putrid character of the empyema. Their mode of access to the pleura is usually through the bronchi, through the gastro-intestinal tract, or an external wound. None of these etiologic factors may be determinable upon the post-mortem table.

Pathology.—The exudate is not always purulent in its gross appearance, and sometimes there is no sharp line of distinction microscopically between a serofibrinous and a purulent exudate, since it is entirely a question as to the number of pus-cells. The fluid may be thin and slightly opaque, or thick and viscid pus. Microscopically, leukocytes, endothelial cells, red corpuscles, large granular cells, fat globules, detritis, cholesterin crystals, and micro-organisms are present.

There are few changes in the pleura and surrounding tissue peculiar to empyema. There is extensive and deeper infiltration of the pleura with round-cells and leukocytes, and the lymphatic spaces are often found engorged with pus, and the

blood-vessels dilated. In long-standing empyema the pleura shows great fibrous thickening, and the lung is compressed. Sooner or later necrosis of the pleura will probably follow, either as the result of pressure or of the local inflammatory process, resulting in perforation of the sac and the discharge of the pus in various directions. The fluid may find its way into the lung, either by soakage or by rupturing into a bronchus, or it may form an external tumor—"*empyema necessitatis.*"

The entire pleural cavity may be affected, or the empyema may be encapsulated between the lung and chest-wall, or in the forms known as diaphragmatic, interlobular, or mediastinal.

Rupture may take place either internally or externally.

Symptoms.—The onset and symptoms of empyema are not typical. The condition may arise insidiously or acutely. In the majority of instances the affection begins insidiously. If it should begin acutely, there will be severe chill, with rapid rise in the temperature, from 102° F. to 105° F., severe pain in the side, and intense dyspnea.

The exudate appears early, and is purulent from the beginning, or it may even be putrid. The constitution of the patient is affected from the onset. There is marked weakness, wasting, depression, dry tongue, with sordes upon the teeth, rapid pulse, and tendency to delirium—in fact, the so-called "typhoid state" is often present. A fatal issue may take place at the end of a week, although these cases are exceptional. Often the onset is insidious, and the course may be absolutely afebrile. The hectic temperature curve may occur, but this is not characteristic. The greater number of cases occupy the middle line between the extreme acute and the very insidious onset. The symptoms may be masked at first by the primary disease, or the disease may develop as a typical serofibrinous pleurisy, with moderate fever, pain, and dyspnea.

As the primary affection declines, the temperature does not fall; on the contrary, in perhaps ten days to two weeks it gradually assumes a hectic type, with chilly sensations and sweating. Accompanying these symptoms there is gradual loss of strength and flesh. The face becomes pale, dyspnea increases, cough is troublesome and dry, and clubbing of the fingers occurs in the older cases, and the urine is albuminous. The patient may succumb from exhaustion, or to secondary amyloid disease, or to some other complication.

Physical Signs.—The physical signs are those of a serofibrinous pleurisy. The character of the fluid gives no characteristic signs. Baccelli thought that he had discovered an important differential sign, in that the voice could be transmitted through serofibrinous exudates but not through purulent ones. This sign has been found to be unreliable, and no dependence must be placed upon it, as the voice-sounds are often equally as well transmitted through purulent as through serofibrinous and hemorrhagic exudates. Displacement of organs in empyema is more apt to be marked than in serofibrinous pleurisy. Local edema and redness of the skin is often present.

Diagnosis of Empyema.—There are no positive signs nor symptoms by which empyema may be diagnosticated from serofibrinous pleurisy.

Fever, sweating, edema, and chills are sometimes present in serofibrinous pleurisy as well as in empyema. Exact diagnosis can only be made upon aspiration.

SPECIAL VARIETIES OF PLEURISY.

Diaphragmatic Pleurisy.—When the inflammation is limited to that portion of the pleura covering the diaphragm and the under surface of the lung, peculiar symptoms may arise. This, however, is not common. It may be secondary to abdominal affections or to diseases of the lung such as lobar pneumonia, or it may be primary, produced by the same causative influences as other forms of pleurisy. The dry variety is exceptional, most cases being accompanied by small effusions, mostly serofibrinous, rarely purulent in character.

The onset of the disease is abrupt, beginning with a chill and a pronounced febrile movement, from 103° F. to 104° F. The pain is extremely severe and referred to the hypochondrium; sometimes it is felt down the back. A pain extending along the course of the tenth rib to the sternum is supposed to be diagnostic.

The face is anxious, the pulse feeble and rapid, and there is excessive dyspnea. The anxiety of the countenance is due to the extreme pain, the patient making every effort to immobilize the chest; he may sit slightly forward with hands placed against the sides of the thorax, or may occupy a semirecumbent position, with elevated knees. The respiration is rapid, costal, and superficial. The abdominal wall upon the affected side is

tense, with firmly contracted muscles, and sensitive to pressure. Pain may also be present at other parts of the chest, both anteriorly and posteriorly, and may be the result of neuritis of the phrenic nerve from pressure, which may persist even after the pleurisy has disappeared.

Pain is increased by movement, by cough, and hiccup, which is occasionally a troublesome symptom. Vomiting frequently takes place, greatly aggravating the pain, and often leading to false diagnosis. In the severer forms delirium is frequent and may be the forerunner of a fatal coma. The bowels are constipated.

Physical Signs.—The physical signs of pleurisy of this form are vague or may be absent altogether. Occasionally friction sounds may be heard at the base of the chest, but on account of the great pain and the slight efforts made in respiration, and their location, they are rarely heard.

When effusion is formed the characteristic zone of flatness and some displacement of the liver and spleen may also be present.

The disease is often mistaken for peritonitis and other grave abdominal affections. In pleurisy the abdomen is not distended, the pain is superficial, and the dyspnea is a marked symptom. If friction is present, it aids materially in the diagnosis. The prognosis of uncomplicated cases is almost always favorable, fatal cases being due to complications and to a purulent effusion.

Hemorrhagic Pleurisy.—The hemorrhagic variety occurs in tuberculosis of the pleura, and is due to degenerative changes of the walls of the blood-vessels.

Hemorrhagic effusions also result from malignant growths of the pleura. The diagnosis can only be made by exploratory puncture.

Chylous Pleurisy.—This form is of exceeding rarity. It may be due to obstruction and rupture of the thoracic duct and from cancerous thrombosis of the subclavian and jugular veins.

Pulsating Pleurisy.—In a left-sided pleural effusion, a synchronous pulsation may occur with that of the heart. This may be noticed in several intercostal spaces upon the affected side. The condition is most apt to be an empyema, and is rare. The pulsation is situated most often anteriorly between the second and the sixth ribs upon the left side, and often forms a tumor; there may even be two or three.

Pulsation posteriorly is very rare. The pulsation may be increased by the patient lying upon the unaffected side.

The prognosis is grave. Pneumothorax may be present. This may be due to the gas production of the bacillus aërogenes capsulatus.

Encapsulated Pleurisy.—When effusions are limited by costopulmonary adhesions, the condition is spoken of as "encapsulated or encysted pleurisy." It is sometimes called "interlobular" if the condition occurs between the two lobes of the lung, or "mediastinal" if between the lung and the mediastinum. In the two latter instances the condition is almost invariably purulent.

The diagnosis is often impossible without exploratory puncture.

When the purulent fluid passes into the lung by an extended surface, by soakage, or by a fistulous tract communicating with a bronchus, it may find its way out through an intercostal space and form a tumor beneath the skin; it is then called "empyema necessitatis."

Pleurisy at the Extremes of Age.—In the aged the condition most frequently occurs as a complication of pneumonia, cancer, or cardiac and renal disease. Pain and fever are mostly absent, dyspnea is moderate, and the course of the disease is insidious. In very young children the effusion is very apt to be purulent.

Complications.—Sudden death may take place during the first weeks of the rapidly appearing effusion, or even later during convalescence. It may be due to syncope, from fatty degeneration of the heart, or undue pressure upon it. Thrombosis of the pulmonary artery and right heart or associated pericarditis may cause sudden death. Fatal syncope may happen during aspiration. Pulmonary edema may also give rise to sudden death. Peritonitis occurs as a complication. Chronic interstitial pneumonia may follow. Gangrene may result from fetid pus penetrating the lung. Pneumothorax may be associated with pleural effusion in consequence of rupture of the lung, or the empyema may rupture into the bronchus. Nephritis sometimes occurs as a complication. There may be clubbing of the finger-tips. Lardaceous or amyloid disease is a rare complication.

Diagnosis.—In simple acute pleurisy with the presence of localized pain and a well-defined friction sound the diagnosis

is easy. The condition may be mistaken for muscular rheumatism, especially of the intercostal muscles. Muscular rheumatism is bilateral; the muscles are painful upon pressure; movement also produces pain.

Intercostal neuralgia sometimes gives rise to difficulty in diagnosis, but the locality of the pain, as also the presence of herpes zoster, will help to differentiate the condition. Gastralgia, ulceration of the stomach, and peritonitis occasionally give rise to difficulty in diagnosis, but the presence of the friction sounds is of service in differentiating the conditions.

Pleural effusions may be mistaken for croupous pneumonia. Under the description of croupous pneumonia the differential table between these two diseases is given. (See p. 236.) Mediastinitis and tumors of the pleura are exceedingly difficult to differentiate from pleural effusion. An important point in favor of pleural effusion would be the absence of vocal fremitus, while in the other condition the fremitus may be preserved; besides, in tumors there are symptoms of pressure upon the nerves, blood-vessels, and esophagus. These signs would be more in favor of tumors than pleural exudation. In all doubtful cases aspiration should be performed.

Pulsating empyema may be mistaken for aneurysm, but in empyema there is absence of thrill and bruit over the pulsating mass; besides, the pulsation in empyema can hardly be spoken of as expansile. Empyema necessitatis may give rise to great difficulty in diagnosis, but the association of involvement of the base of the lung of the corresponding side with displacement of organs will serve to make the proper diagnosis.

Hydrothorax, if it be unilateral, may give rise to difficulty in diagnosis. The absence of friction sounds, severe pain, and the subfebrile rise in the temperature are of importance. A large pericardial effusion may be mistaken for a left-sided pleural effusion, but care in the physical examination, with the history, will help to differentiate the conditions.

Prognosis.—Acute fibrinous pleurisy, if it be primary, is almost always favorable. In serofibrinous effusion, the etiologic factor must be taken into consideration, although ultimate recovery most often follows. Empyema is a very serious condition. In pneumococcus infection the mortality is very low, about 2% or 3%, whereas in the streptococcus and mixed infections the mortality reaches 25%. Empyema of tubercular origin is a very chronic process, life being prolonged, and restoration to health may even occur.

Treatment.—Dry Pleurisy.—The indication is to relieve pain. This may be accomplished by the local use of dry cold, or sinapisms. Strapping the chest with strips of adhesive plaster so as to render the affected side immobile is often followed by marked relief. Internally, the analgesic antipyretics are of use, or small doses of Dover's powder or some other form of opium may be administered. If the pain be very severe, hypodermic injections of morphin may be necessary. If the pain continue, it may yield to small fly blisters.

Pleurisy with Effusion.—The fluid must be removed either by absorption or aspiration. There are no positive signs by which the nature of the fluid can be definitely known; however, if pus is supposed to be present, aspiration should be resorted to at once.

Counterirritants, diaphoretics, diuretics, and laxatives may be employed, but are of very little value. It is recommended by some to exclude fluids from the diet, so as to produce absorption.

Should the effusion remain stationary or continue to increase, or if the fluid should reach the level of the second rib, or should there be evidences of grave respiratory and circulatory disturbances, an exploratory puncture should be made at once. In instances where the effusion is complicated with pericarditis or valvular lesions of the heart, immediate aspiration is necessary. When there is septic temperature with development of edema of the chest-wall, a purulent pleurisy should be suspected and exploratory puncture at once made.

The operation of aspiration is not difficult. The chest is prepared with proper antiseptic precautions, and the point of selection for puncture is in the posterior axillary line in the seventh interspace. The patient should sit up, and rest the hand of the affected side upon the opposite shoulder. It is important to test the aspirator before the operation. The needle should be guarded by the fingers so that it does not enter the chest-wall too deeply, and then inserted into the chest just above the border of the rib. In this way the danger of wounding the intercostal artery is averted. The possibility of puncturing the diaphragm, or entering the liver upon the right side and entering the spleen upon the left side, must be remembered. It is well to withdraw the fluid slowly, and the conducting tube should be compressed from time to time for this purpose.

It is not always necessary to withdraw the entire effusion,

especially if it be very large. If urgent dyspnea, great cough, or pain, faintness or syncope, or the appearance of blood in the aspirated fluid occur, the operation should be suspended at once.

After the needle has been withdrawn, the finger should be held over the point of puncture until cotton moistened with collodion or adhesive strips are applied.

The treatment of purulent pleurisy consists in free drainage, with thorough antiseptic precautions. The pus is often so thick that it will not flow through the needle. In such cases a surgical operation is necessary.

CHRONIC PLEURISY.

The disease occurs in two forms—(*a*) pleurisy with effusion; (*b*) dry pleurisy.

(*a*) **Pleurisy with Effusion.**—This may follow the acute form or it may begin insidiously, the pathology being the same as in the acute varieties. If the fluid be absorbed or removed, there is retraction of the affected side, sometimes giving rise to deformity. Subjective symptoms, with perhaps the exception of slight dyspnea, are mostly absent. The pulse may be slightly increased in frequency, and there may be slight evening rise of temperature. If the effusion remains for any time, especially in children, pus is likely to form and hectic temperature and edema of the chest-wall may develop. The physical signs of a pleural effusion are always present. The disease may last for months or years. Death is usually due to some chronic suppurative process or to pulmonary tuberculosis.

(*b*) **Chronic Dry Pleurisy.**—This may follow acute or chronic pleurisy with effusion. If the exudate be absorbed, the layers of the pleura, which still contain some fibrinous material, come together, and subsequently fibrous connective tissue is formed. Usually this appears most prominently at the base, giving rise to flattening and retraction of the chest-wall upon the affected side. From the great thickening of the pleura, impairment of resonance and some enfeeblement of the breath-sounds is commonly noticed. Should the condition have followed empyema, the retraction and flattening will be still greater. In these fibrous bands which stretch across the pleura small cysts may form. Fibrous pleurisy may follow acute fibrinous pleurisy, and adhesion of the layers of the pleura is

apt to result. These adhesions are very commonly found at autopsies. They may be general or limited. Ordinarily, no change upon physical examination is noted, except when the adhesion is general or very thick. There is some impairment to expansion of the affected side, and the breath-sounds may be weaker, although this is by no means the rule. Friction sounds are sometimes heard.

Treatment.—The effusion, if present, should be removed, and the general nutrition of the patient must be looked after. The diet should be generous. Climatotherapy and lung gymnastics are of use. General tonics are especially indicated.

PNEUMOTHORAX.

Definition.—Air in the pleural sac.

The presence of air alone is an exceedingly rare condition. In the majority of cases an effusion, either of a serous or purulent character, is also present, producing a hydropneumothorax, or a pyopneumothorax. When there is blood with air in the pleura cavity, the condition is known as hemopneumothorax.

Synonyms.—Hemopneumothorax ; pyopneumothorax.

Etiology.—This disease may arise from two sources, first, by perforation—(*a*) external perforation, (*b*) internal perforation ; second, by spontaneous development of gas within the sac.

1 (*a*) *External Perforation.*—Trauma, stab wounds, gunshot wounds, blows, by means of the aspirator, operation upon the chest, and caries of the ribs. (*b*) *Internal Perforation.*—The most common instances of this form of perforation are : In case of a tubercular cavity, this ulcerating through the visceral pleura ; from caseous tuberculosis without cavity formation, a common communication being established with a bronchus ; abscess of the lung and gangrene ; infected emboli, causing ulceration of the inner layer ; chronic bronchopneumonia ; emphysema ; and from an empyema, causing a perforation into a bronchus. Rarely, from whooping-cough, a rupture occurs internally. Hydatid and carcinoma have caused the condition.

Sometimes the perforation is through the diaphragm, as from a subphrenic abscess which communicates with a perforated gastric ulcer.

2. In some instances pneumothorax is believed by many to

arise from gas production due to the bacillus aërogenes capsulatus; this, however, is doubted by some.

The disease is more frequent in males than females, and occurs most often upon the left side of the chest.

Pathology.—It can be readily understood that from an external injury air may gain entrance into the pleural cavity. Infection frequently follows. The quantity of air that may find entrance into the pleural cavity varies greatly in amount. Displacement of adjacent organs depends upon the distention of the pleural sac. The air may be absorbed.

Symptoms.—Under ordinary circumstances, with a rupture of the pleura sudden distressing pain is the most important symptom. It may occur during an attack of coughing, and be followed by great difficulty in breathing. The condition, however, may come on insidiously without pain, cough, or dyspnea.

As a rule, the pulse and respiration are greatly increased in frequency. Cyanosis is prominent, the expression is anxious, the alæ of the nose moving, and the auxiliary muscles of respiration called into activity. The extremities become cold; the temperature is lowered. The patient may be bathed in cold perspiration; in fact, all the symptoms of collapse are present.

The dyspnea, which is perhaps the most important symptom, is often extreme. The decubitus varies; the dyspnea may be so great that the patient must be propped up in bed; commonly he is found lying upon the affected side.

Physical Signs.—Inspection.—The shoulder upon the affected side is elevated, the intercostal spaces being partially or completely obliterated, the side distended, and respiratory movements diminished or completely absent. Respiration upon the unaffected side is increased.

The impulse of the heart is displaced toward the healthy side, and on account of the rapid and disturbed respiratory excursis or the weakness of the heart's action great difficulty will be found in locating the impulse exactly.

Palpation. — Palpation confirms inspection. The vocal fremitus is absent. Downward displacement of the liver, if the affection takes place upon the right side, will be noted.

Percussion.—The signs upon percussion are characteristic. A tympanitic note is noticed high up in the chest above the level of the fluid. Below where the fluid accumulates, flatness is noted upon percussion. *The level of tympany and*

flatness varies with the changed position of the patient. If the condition be upon the left side, Traube's semilunar space is found displaced.

Auscultation. — Auscultation shows absence of breath-sounds upon the affected side. Occasionally rales of a distinct metallic quality may be found present (metallic tinkling ; gutta cadens). Vocal resonance is absent. The characteristic sign is the succussion splash, which is due to the association of air and fluid in the pleural cavity. The sound is elicited by placing the ear to the affected side and vigorously shaking the patient. It often has the metallic quality. The patient may be conscious of this sound himself.

The coin test is elicited in this affection. By placing a coin upon the diseased side anteriorly, and tapping with another coin, the examiner listening posteriorly, a ringing, metallic, bell-like sound is transmitted to the ear. This does not occur upon the healthy side. This sign may be heard in any large cavity with tense, fine walls.

Diagnosis.—The diagnosis depends upon the history, the pain with dyspnea, cyanosis, fall in temperature, and the association of characteristic physical signs, especially the succussion sound, with displacement of organs.

Prognosis.—The prognosis depends upon the cause. In simple traumatic cases the opening often becomes sealed by inflammatory exudation, the air being absorbed. In tuberculous cases the prognosis is bad.

Treatment.—The treatment is symptomatic and palliative. Opium and stimulants are particularly useful. Paracentesis must be considered when the effusion becomes marked. For the pain, strapping of the affected side is of advantage in some cases. It is necessary to know the nature of the fluid present, and if it is found to be pus surgical interference is necessary.

HYDROTHORAX.

Definition.—An accumulation of fluid, without inflammatory signs, in the pleural cavity.

Etiology.—This is always a secondary affection, and occurs commonly in chronic disease of the kidneys and heart, and anemic states. It may occur from the pressure of tumors, and is often the terminal event in many acute and chronic diseases.

Pathology.—The disease is most often bilateral, although the fluid frequently is greater upon one side than the other.

It may vary from a small amount to several liters. In diseases of the heart, one pleural cavity alone may be affected. If both are affected, there is often a much greater amount of fluid upon one side than upon the other. The fluid is clear, straw-colored, and of low specific gravity—below 1018. Inflammation or roughening of the pleura is not present.

Symptoms.—The symptoms of the primary disease are always most marked. When transudation into the pleura takes place, dyspnea becomes a prominent symptom. It may be associated with cyanosis, great distress and anxiety, and profuse cold perspiration. Pain is absent, and cough is not constant.

Physical Signs.—The physical signs are those of a pleural effusion without the early presence of friction sounds. If the disease be bilateral, displacement of organs is not a striking feature.

Diagnosis.—The condition must be differentiated from pleural exudations.

Differential Diagnosis.—

Hydrothorax.	*Pleural Effusion.*
The disease is bilateral.	The disease is unilateral.
No pain; cough not marked; always secondary to other diseases.	Pain; cough; often a primary affection.
Friction sound absent.	Friction sound present.
No displacements of organs apparent.	Displacement of organs common.
No fever.	Slight fever.

Treatment.—The treatment is that of dropsy appearing in other parts of the body. The fluid may be removed by aspiration. Attention should be directed to the primary disease.

HEMOTHORAX.

Definition.—Blood in the pleural sac.

Etiology.—Hemothorax should not be confounded with hemorrhagic effusion. It may result from traumatism that causes fracture of a rib or wounding of a lung. It may also result from the rupture of an aneurysm, from malignant diseases of the lung, and hemorrhagic diathesis.

Symptoms.—The symptoms are those of hydrothorax, with those of blood loss. Pallor, a small and sometimes imperceptible pulse, great dyspnea (air hunger), and decrease in the quantity of urine are present. There may or may not be pain.

Physical Signs.—The physical signs are those of a pleural effusion without the friction sound.

Prognosis.—Traumatic cases may result in cure. The condition, resulting from any cause, is grave.

Treatment.—Hemothorax, if it be moderate in size, should not be interfered with, as subsequent absorption and clotting may take place. Rest and the free administration of opium are necessary; avoid stimulation, for it will interfere with the formation of the clot.

TUMORS OF THE PLEURA.

Benign Tumors.—Of the benign connective-tissue tumors, fibromata and lipomata have been observed, the latter frequently appearing upon the costal pleura.

Malignant Tumors.—These are rare primary affections. Carcinomata invade the pleura by extension, this being either from the lungs, mediastinum, or from the mammary gland, rarely from more distant growths. The tumors are usually small. Enlargement of the cervical lymphatic glands is often present.

Sarcomata may be primary, either springing from the endothelial layer (the endotheliomata) or from the subendothelial connective tissues. The sarcoma is frequently of the spindle cell variety. Secondary involvement of the lungs often occurs. G. R. Butler ("New York Medical Journal," 1895) collected seven cases from literature of primary sarcoma of the pleura, of which six affected the left side. Sarcoma may also involve the pleura secondarily.

Symptoms.—There are no characteristic phenomena which show the development of malignant diseases of the pleura. Dull, persistent pain in the affected side, with great weakness and dyspnea, and signs of pleural effusion of a hemorrhagic form are often present. Some degree of cachexia is noticeable, and the lymph-glands, especially of the neck and axilla, are frequently found enlarged if the growth be carcinomatous. If the tumor be very large, bulging of the chest may result upon the affected side. In the endotheliomata there is retraction, with dullness or flatness upon percussion, and absence of respiration over the affected side. The fluid present may obscure the signs of the malignant growth. The most important signs depend upon the exploratory puncture.

The following rules have been laid down by Fränkel: The deep-red color of the fluid, almost like that of venous

blood, the discovery upon microscopic examination of small particles of the growth itself, which show its organic structure, the presence of considerable fat, either free, as a chylous fluid, or inclosed in epithelial cells, are important factors.

Prognosis.—The prognosis is grave.

Treatment.—The treatment is palliative.

ECHINOCOCCUS OF THE PLEURA.

This is a very rare condition. It is most often secondary, but may be primary. Davaine (Traite des Entozoaires, Paris, 1860) reported twenty-five cases of hydatid cysts of the pleura, only one of which he believed to be primary. In the secondary form either the liver or the lung is the organ first affected. The cyst is most often single and sterile, but not invariably so. The disease may affect the pleura from the rupture of a hepatic cyst. The contents of the cyst may be either clear or purulent.

Symptoms.—The onset is insidious; occasionally there may be sharp or sudden pain. In any event, sooner or later pain becomes a prominent symptom, and is very persistent. As the cyst develops, dyspnea and cough appear.

Physical Signs.—The physical signs are those of an effusion. There may be circumscribed bulging of the affected area. On puncture, a fluid is obtained in which hooklets may be discerned by the aid of the microscope.

Prognosis.—The prognosis is serious, but not so grave as when found in the lung.

Treatment.—Surgical interference is necessary, and aspiration may be performed.

DISEASES OF THE MEDIASTINUM.

INFLAMMATION OF THE MEDIASTINUM.

Synonym.—Mediastinitis.

Inflammation of the mediastinum may be simple or infective, acute or chronic, primary or secondary. Simple acute inflammation which terminates in resolution can not be diagnosticated.

Etiology.—This condition may occur by extension from chronic pericarditis, chronic inflammation of the pleura, chronic

peritonitis, and chronic inflammation of the tracheobronchial glands.

Pathology.—A fibrinous exudate, later the development of fibrous tissue in the shape of dense bands surrounding the blood-vessels, trachea, and bronchi, often forms. The tissues undergo consolidation from contraction of the fibrous bands, producing pressure effects upon the veins, the aorta, the nerves, and may frequently be great enough to constrict the trachea and some of the larger bronchi.

Symptoms. — The symptoms of this disease are often obscured and masked owing to the primary condition which precedes the disease, such as the inflammation of the pericardium, pleura, bronchi, and lung tissue. The disease may be diagnosticated from an increased fullness in the jugular veins, which steadily increases. This will be especially noted during inspiration, and lessened in expiration, the pulsus paradoxus being associated with the condition. Pain is present behind the sternum, which may radiate to the shoulders, back, neck, or chest. Dyspnea, increased upon slight exertion, is common. The pulse is rapid, small, easily compressed, and of low tension, besides having the character of the pulsus paradoxus. Cough with expectoration, followed later by cyanosis, is a common symptom. If constriction of the inferior vena cava occurs there will be swelling of the liver, ascites, and dropsy of the lower extremities. Fever may or may not be present.

Physical Signs.—As a rule, there is an increase in the area of precordial dullness; the apex-beat of the heart is absent, the sounds being faint though regular.

Diagnosis.—This is extremely difficult, and must often be made by exclusion.

Prognosis.—The prognosis is unfavorable.

Treatment.—The treatment is palliative, symptomatic, and supporting.

ABSCESS OF THE MEDIASTINUM.

Etiology.—All the causes which have been mentioned under inflammation of the mediastinum may give rise to abscess, provided some pyogenic micro-organism be present. The streptococcus infection produces a severe spreading variety, the staphylococcus infection, as a rule, giving rise to the milder, more circumscribed forms. Trauma is a frequent cause. The

condition may result from actinomycosis, from erysipelas, pyemia, variola, scarlet fever, measles, enteric and typhus fever, and from other infectious diseases. The disease occurs more frequently in males than in females.

Pathology.—The seat of abscess is most frequently in the anterior mediastinum. The pathology is that of abscess; the pus finds its way between various organs, and some of these may be perforated. If the infection be of a slow or chronic nature the abscess wall may be quite thick. Various forms of micro-organisms have been isolated from the pus.

Symptoms.—If the abscess be acute, the ordinary signs of purulent inflammation are present, such as chill, fever, sweating, anorexia, coated tongue, vomiting, constipation which is followed by diarrhea, loss of flesh, anemia, great depression, and muscular weakness. Dry spasmodic cough, later accompanied by expectoration, which is often purulent and blood-stained, is a symptom. The local manifestations are indefinite in character; there may be a sense of fullness and constriction behind the sternum, which may give place to a constant dull, aching pain. Tenderness upon pressure is present over the sternum. If pressure upon the veins take place, signs of venous engorgement are noticed, and in pressure over the vena cava ascites and edema of the lower extremities result. Pressure upon the great veins gives rise to murmurs, and pressure upon the esophagus results in dysphagia. If there is pressure upon the laryngeal nerves, paralysis of the vocal cords is noted. The physical signs vary, depending upon the location of the abscess. The heart may be displaced; the sounds are apt to be obscured but regular. The pulsus paradoxus is likely to be present.

Prognosis.—If the abscess can be evacuated the prognosis is more favorable than in the deep-seated cases. Under any circumstance it is a very serious affection.

Treatment.—The treatment is surgical.

TUMORS OF THE MEDIASTINUM.

BENIGN TUMORS.

Lipomata, fibromata, enchondromata and osteomata rarely occur in the mediastinum. Dermoid and echinococcus cysts have been encountered in this locality.

MALIGNANT TUMORS.

Carcinoma of the Mediastinum.—This tumor occurs more commonly as a primary growth than as a secondary, being most common in the anterior mediastinum. They occur most frequently between the ages of fifty and sixty.

Sarcoma of the Mediastinum.—The most common seat of this tumor is also in the anterior mediastinum, it being more frequently primary than secondary.

Secondary carcinoma also occurs in the mediastinum.

Symptoms.—Tumors of fairly large size soon produce symptoms of pressure upon the heart or its vessels, the lung, the recurrent laryngeal nerve, the trachea, the bronchus, the large blood-vessels, such as the superior or inferior vena cava, the esophagus, and the sympathetic nerves. The symptoms are those of dyspnea, cough, frequently of an aneurysmal character, difficulty in swallowing, variation in the size of the vessels, and other circulatory disturbances. Cyanosis and enlargement of the thoracic veins arise due to the attempt at collateral circulation.

The physical signs are varied. If the tumor be very large, upon inspection there may be bulging of the sternum and ribs, and these bones may become eroded. If the growth push the heart forward, marked pulsation may be noted. Upon inspection, slight cyanosis and edema of the superficial veins may also be apparent. Upon palpation the vocal fremitus is found to be greatly decreased or absent. Upon percussion, dullness merging into flatness is present over the affected area. Upon auscultation indistinct or absent breath-sounds, with decreased or absent vocal resonance, is noted. If the tumor be situated anteriorly to the heart, the apex-beat will be faintly visible or absent, and the heart-sounds upon auscultation will be distant and muffled. If the tumor be carcinomatous in nature, enlargement of the axillary and cervical lymphatic glands will be noted if these have become invaded by the spread of the growth.

Prognosis.—The prognosis is grave.

PART IV.

DISEASES OF THE DIGESTIVE TRACT.

DISEASES OF THE MOUTH.

CATARRHAL STOMATITIS.

Definition.—An inflammation of the mucous membrane of the mouth.

Synonyms.—Acute stomatitis; simple stomatitis.

Etiology.—The disease is most frequent in children, but may also occur in adults. The inflammation appears in the form of erythematous patches, so that the surface is drier than normal; or a catarrhal inflammation develops, with some thickening and increased secretion.

Improper food, either too hot or pungent, prolonged sucking of an ill-developed nipple, or unclean feeding bottles are causes. Dentition may be a cause; and in adults, tobacco, stimulating food, alcohol, or an irritation from a carious tooth.

Some drugs, such as mercury, arsenic, lead, iodin or bromin, may cause the disease. It may be the result of gastro-intestinal disturbance, and occurs in some of the specific fevers, such as scarlet fever and measles, rarely in tuberculosis, carcinoma, and diabetes.

Symptoms.—The symptoms are pain, heat, discomfort, dryness of the mouth, and difficulty in swallowing. The child is fretful, saliva drivels at the mouth, and the patient may become wasted and feeble. There is usually slight fever, accompanied by vomiting and diarrhea.

Treatment.—The treatment consists in cleanliness, careful feeding, and the use of mild alkaline mouth-washes. If pain be great, ice may be used, or a weak solution of cocain painted upon the erythematous patches. A mild purge is often advantageous.

ULCERATIVE STOMATITIS.

Synonym.—Putrid sore mouth.

Etiology.—The disease most frequently occurs in children between the ages of four and sixteen, and is usually due to bad sanitary surroundings, or a local irritation such as a decayed or carious tooth. Improper feeding may give rise to the affection.

Pathology.—The pathology consists in a localized necrosis of the buccal mucous membrane with surrounding inflammatory infiltration. The ulceration spreads superficially.

Symptoms.—The disease commonly appears first at the margin of the gum in the region of a molar tooth, oftener on the left than on the right side. The gum is red and swollen, and bleeds easily. The mucous membrane soon ulcerates, leaving a dirty yellow or grayish ulcer. The cheek and tongue upon the implicated side are also affected, and the ulceration may spread to the lips.

The breath is offensive, and the lymphatic glands of the neck are enlarged. The tongue is thickly coated. The duration of the disease depends upon the extent of severity of the affection.

Treatment.—Alkaline washes for the mouth, or peroxid of hydrogen in small doses, are effective. Chlorate of potash, locally, has been recommended by some authorities.

APHTHOUS STOMATITIS.

Synonyms.—Aphthæ; vesicular stomatitis.

Etiology.—The affection occurs most frequently in children, and the usual causes which have been indicated in other forms of stomatitis are also prominent here. The disease occurs occasionally in adults, in whom it is most likely due to some local irritation.

Symptoms.—Small papules, either single or in groups, occur in parts of the mouth, most commonly upon the inner surface of the lower lip. At the end of twenty-four hours the epithelial covering is lost, and a small oval, whitish patch, with raised edges, makes its appearance. This disappears in a day or two and leaves a small conical ulcer behind. Pus formation does not take place. The sore heals in a few days,

new epithelium covering the ulcer. The duration of the disease is about seven days.

Treatment.—Constitutional difficulties must be carefully inquired into. The mouth should be washed before food is given, and weak alkaline solutions of boracic acid, five grains to the ounce, or a 1% solution of permanganate of potash may be painted upon the spots. If there be great pain, a weak solution of cocain may be applied from time to time. A mild purge should be given at the onset.

PARASITIC STOMATITIS.

Definition.—The disease is a catarrhal stomatitis, associated with a large fungous growth, consisting of white patches of various sizes. The back of the tongue and the inner surface of the cheeks and palate show the affection most prominently. It is generally believed that the cause is the fungus known as the oidium albicans.

Synonyms.—Thrush; muguet.

Etiology.—Uncleanliness, constitutional debility, and coexisting catarrhal stomatitis are predisposing factors. It occurs most frequently in children, and next commonly in old age. It is supposed that restricted movement of the tongue, which permits the lodgment and growth of the fungus, has something to do with the etiology. The acid condition of the secretions of the mouth, which seem to favor its growth, are probably a result, and not a cause. Unclean feeding bottles, spoons, etc., are sources of infection.

Pathology.—The oidium albicans lodges upon the perfectly normal mucous membrane. Occasionally a catarrhal condition of the mucous membrane may precede the parasitic growth. The parasite is simply confined to the superficial epithelium, and may be seen upon the tongue, cheeks, lips, hard palate, or the tonsils, as white spots, which are slightly raised, and may increase in size by extension.

Symptoms.—The symptoms are slight. The disease is noticed by an inspection of the mouth. A catarrhal stomatitis is always associated. There may occasionally be diarrhea and vomiting.

Diagnosis.—Small particles of the adherent curd-like patch, when subject to microscopic examination, reveal the fungus.

Prognosis.—The prognosis is favorable.

Treatment.—The treatment consists in cleanliness. If the

child be not breast fed, a wet-nurse should be supplied. The fungus may be wiped off with soft linen soaked in boric acid solutions and solutions of carbonate of sodium, or permanganate of potassium may be of use. In cachectic individuals alcohol and tonics are necessary.

MERCURIAL STOMATITIS.

Definition.—An inflammation of the buccal mucous membrane and salivary glands resulting from the administration of mercury.

Synonym.—Ptyalism.

Etiology.—It frequently occurs in individuals who are especially susceptible to small doses of the drug, or it may arise from an excessive administration. Occupations in which the drug is handled may predispose. Any of the preparations of mercury may cause stomatitis. Small doses of calomel, frequently repeated, will very often produce the condition. When mercury, especially in the form of calomel, is administered, it is wise to ask the patient whether he is subject to ptyalism.

Symptoms.—The early symptoms are swelling of the gums, accompanied by tenderness and some pain, especially marked on mastication. The breath in this condition is offensive. Accompanying these changes, the saliva increases in amount and may be excessive. The teeth may become loose; ulceration of the gums and, rarely, necrosis of the jaw may follow. The inflammation may spread to the pharynx and Eustachian tubes. When administering mercury, tenderness of the gums is suggestive of beginning ptyalism. A metallic taste in the mouth may sometimes be noticed. Diarrhea and abdominal pains are likely to be accompaning symptoms.

Prognosis.—This disease is rarely of a serious nature; sometimes, however, it is very distressing.

Treatment.—The administration of mercury should be suspended. Mouth-washes, such as chlorate of potassium and listerine, are useful, and frequently the only remedies necessary. Doses of atropin may be beneficial. The bowels should be freely opened with salines. Iodid of potassium is sometimes useful. If pain exists, opium should be administered.

GANGRENOUS STOMATITIS.

Definition.—A disease characterized by extensive destruction of the cheek.

Synonyms.—Noma; cancrum oris.

Etiology.—This is a rare form of stomatitis, usually occurring in persons in bad health, or those subject to unsanitary surroundings, starvation, malaria, or other acute and chronic diseases. In children the disease often follows measles, and sometimes enteric fever. It occurs most frequently in damp countries. It is a disease of children between the ages of two and five.

Pathology.—The first lesion is usually upon the mucous surface of one of the cheeks, appearing as a small bleb, followed by ulceration and induration of the surrounding tissues, which rapidly spreads over the whole cheek, the area becoming gangrenous. The area of disease is tense, may be red and in some instances black, and may extend to the upper or lower jaw, the malar bone, and sometimes the orbit. Perforation of the cheek not infrequently occurs. The teeth may become loose and drop out. The blood-vessels show the greatest resistance, but hemorrhage does not take place. If the condition terminates favorably, a large and extensive cicatrix will result, causing great deformity.

Symptoms.—One of the earliest symptoms is a foul odor from the mouth, with redness and swelling of the cheek. Pain is not usually present even when examination of the mouth reveals a sloughing ulcer, nor even when the bone is attacked. Extreme prostration, with delirium, occurs, especially in cases terminating fatally. Bronchopneumonia is very apt to take place. The fever is moderate, pulse is rapid, and only slight swelling of the lymphatic glands occurs. The disease usually appears on one side, it being extremely rare for both cheeks to be affected. Diarrhea is present, and often actual ulcerative colitis. Gangrene may occur in other parts of the body, such as the lungs, limbs, or the genital organs.

Prognosis.—Death ensues in about 80% of the cases. The prognosis depends largely upon the extent and rapidity of the disease. With recovery, there is great deformity of the cheek and eversion of the lower eyelid.

Treatment.—The treatment is surgical. The entire mass of gangrenous material should be excised, either by the knife

or the actual cautery. In anesthesia, great care must be taken that none of the material is swallowed or gains entrance into the trachea, as almost certain bronchopneumonia will result. Nitric acid is also used to limit the extent of the disease. The strength of the patient should be maintained. Liquid nourishment in a concentrated form at frequent intervals, and free stimulation, is necessary. Complications must be treated as they arise.

DISEASES OF THE TONGUE.

ACUTE GLOSSITIS.

Definition.—An acute inflammation of the tongue.

Etiology.—Exposure to cold, especially in alcoholics, is a frequent cause. Injury from biting the tongue, as in an epileptic paroxysm or the acute specific diseases, may give rise to the condition. Occasionally only one-half of the tongue may be affected (hemiglossitis). The disease occurs more commonly in men than in women.

Symptoms.—Pain is a most prominent symptom. The tongue is swollen and increased in size, the surface is dry, and the breath offensive. The condition may last two or three days.

Treatment.—A mild saline purge, followed by antiseptic mouth-washes, is usually of benefit. If the pain be severe, painting with a weak solution of cocain will give good results.

CHRONIC GLOSSITIS.

Etiology.—This condition may be either superficial or deep. It may be due to friction from a broken tooth or any irritant. It is frequently the result of alcoholism and dyspepsia.

Symptoms.—The surface of the tongue is smooth and glossy. Parts of the tongue are red- and raw-looking, whereas elsewhere it may be white. Occasionally ulceration may be noted. In severe cases the tongue is swollen, and marked by the edges of the teeth. Pain and stiffness of the organ may be present, aggravated by speaking and eating.

Prognosis.—This condition may predispose to epithelioma.

Treatment.—The diet of the patient should be regulated;

food should be plain and wholesome. Alcohol and tobacco must be avoided. The teeth should be carefully looked after. Dyspepsia and constipation must be corrected. Mild alkaline washes are useful. The ulcers should be painted with a weak solution of nitrate of silver.

DISEASES OF THE SALIVARY GLANDS.

HYPERSECRETION.

Synonym.—Ptyalism.

Etiology.—This condition results from the administration of certain drugs, such as mercury particularly, iodin compounds, gold, copper, jaborandi, and from the use of tobacco. It may accompany inflammatory conditions of the mouth, and sometimes the infectious diseases, such as smallpox. Diseases of the pancreas may be accompanied by hypersecretion. During pregnancy the condition may occur. It is sometimes of nervous origin.

Treatment.—If possible, the exciting cause should be removed, and the condition should be treated the same as mercurial ptyalism.

XEROSTOMA.

Definition.—This is a rare malady, and is associated with nervous disturbances, and is most frequently met with in women. The tongue and buccal mucous membrane become dry and sometimes fissured. Mastication, deglutition, and articulation are impaired. The condition is sometimes associated with diabetes.

Synonyms.—Dry mouth; arrest of the salivary and buccal secretion.

Treatment.—The patient may secure relief by frequent use of water or oil. Occasionally pilocarpin, internally, is of benefit.

INFLAMMATIONS OF THE SALIVARY GLANDS.

Parotitis.—There are two varieties, *specific parotitis*, or *mumps* (already described among the infectious diseases—see

page 261), and *symptomatic parotitis;* the latter form may be acute or chronic, and is known as *parotid bubo.*

Parotid Bubo.—Parotid bubo occurs as a complication in a great many of the infectious diseases, such as enteric fever, typhus, septic disease, tuberculosis, and pneumonia. It also appears in malignant diseases. It occurs in genital affections, such as orchitis and epididymitis, and in inflammation of the ovary. The parotid gland is swollen, and the affection is apt to be unilateral.

Suppuration takes place in a number of the cases. The occurrence of parotid bubo in the course of an infectious disease is considered unfavorable.

Chronic Parotitis. — This condition may follow specific parotitis, or inflammations of the throat. Lead, mercury, and chronic Bright's disease are also etiologic factors. Swelling and tenderness of the glands is present, and there may be hypersecretion. The disease may persist for years.

Treatment.—Ice may be used, or ointments of iodin or mercury to favor resolution and prevent pus formation. Leeches are also beneficial. When pus is present speedy evacuation is necessary. Flaxseed poultices frequently give great relief.

DISEASES OF THE TONSILS.

ACUTE TONSILLITIS.

(*a*) *Follicular or lacunar tonsillitis;* (*b*) *suppurative tonsillitis or quinsy.*

ACUTE FOLLICULAR TONSILLITIS.

Synonym.—Acute amygdalitis.

Etiology.—Young adults are most often affected. In children the chronic form, in which exacerbations take place from time to time, is an important factor. Exposure predisposes. Both sexes are equally liable. The disease appears most frequently in the Spring and Autumn; one attack predisposes to others. It occurs epidemically. Acute rheumatic fever is often associated with the disease. In scarlet fever acute tonsillitis is often a complication.

Pathology.—One or both tonsils may be affected. The tonsils first become swollen and red; very soon the lacunæ are filled with an exudate, which is white, and of a cheesy

consistence. These points of exudation may project from the surface. A number of these areas are frequently found, which may coalesce.

Microscopically, the changes which are found in acute inflammations are present. The exudate consists largely of fibrin, leukocytes, epithelial cells, and various bacteria, such as the staphylococcus and streptococcus, the latter being the one most frequently associated. When the exudate is removed the surface of the involved tonsil does not bleed.

Endocarditis and pericarditis have been found associated with it. Marked leukocytosis is present. The condition may terminate in the suppurative variety.

Symptoms.—Occasionally the constitutional signs are slight, but in the majority of instances there is lassitude and malaise, and in severe cases prostration and great fatigue upon muscular or mental effort. Headache and pain in the bones, which may be violent and increase for some days, are present. Delirium may occur in children. The tongue is coated and the appetite is poor. Vomiting may take place, and constipation is the rule. Fever is almost invariably present; it often reaches 102° F., and not infrequently 104° F. or 105° F., especially in children. Chilly sensations may be present at the onset, and occasionally a genuine chill. The pulse is rapid and the respirations are increased. The fever range is not typical. The urine shows the changes common in fevers; occasionally there is a trace of albumin, and a few hyalin, or granular casts, are present. The symptoms of the general infective process are out of proportion to the local process. This is especially true when the disease occurs in children.

Tickling and burning sensations in the throat, with pain in swallowing, are the early symptoms. Salivation may occur. Muscular movement, especially of the head and neck, is painful. The cervical glands may be enlarged. The pain in swallowing may radiate to the ear. Examination of the throat shows redness and swelling of one or both of the tonsils, with yellowish-white patches upon them varying in size from a pinhead to a split pea. These may be stripped off, leaving an intact mucous membrane beneath. The breath may be foul. At the height of the disease, murmurs may be heard over the base of the heart. It must be borne in mind that true endocarditis is sometimes met with in the course of this affection, and that the murmurs present are not necessarily hemic in origin.

Differential Diagnosis.—

Follicular Tonsillitis.	*Diphtheria.*
Exudate limited to the tonsils.	The exudate upon the pharynx, uvula, and tonsils.
The exudate occurs in the follicles.	The exudate is extensive.
The exudate strips off with ease, but no bleeding surface left beneath.	The exudate is stripped off with difficulty, leaving bleeding surface and probably reforming.
Streptococci frequently found in the exudate.	Klebs-Löffler bacillus present.

Course and Prognosis.—The acute symptoms usually disappear in from two to three days. The prognosis is favorable.

Treatment.—The disease is self-limited and can not be aborted. The patient should be put to bed and a laxative administered. The coal-tar products, especially phenacetin, in small doses, guardedly given, promptly relieve the pain. Quinin in large doses is useless. Local treatment, as a rule, is not of much benefit. Ice bandages and small particles of ice in the mouth are useful. Liquid nourishment should be given, preferably in the form of cold drinks. If pain and sleeplessness are present, opium, especially Dover's powder, may be administered.

SUPPURATIVE TONSILLITIS.

Synonyms.—Quinsy; abscess of the tonsil.

Etiology.—The disease occurs most frequently in males between the ages of twenty and forty, being more common in the spring and late autumn. Certain families show a marked liability to the disease; one attack predisposing to others. Many writers regard it as in some way associated with acute rheumatic fever. Persons with chronic hypertrophied tonsils are more apt to suffer from quinsy than from the follicular form. The attack is preceded usually by exposure to cold, or one of the eruptive diseases, or by a preceding attack of follicular tonsillitis.

Pathology.—It may be unilateral or bilateral. The tonsils become swollen, red, and edematous, and sometimes the surrounding structures are also inflamed. Abscess formation frequently results, and rupture may occur externally—this being the more common—or internally. The carotid artery has been known to rupture, due to an extension by the inflammation.

Symptoms.—The disease most often begins with a severe chill, followed by high temperature (104° F. or 105° F.),

with severe pains in the back and limbs, headache, and constipation. The pulse is rapid, and the local distress, especially when both tonsils are involved, is enormous. Sleep and relief of pain is often impossible without the use of opiates. The pain in the throat is severe, the mouth being opened only with great difficulty. It is not confined to the tonsil, but may shoot to the ear, the floor of the mouth, or to the angle of the jaw. It is deep seated, gnawing, and often neuralgic in character. Salivation is present, the secretions of the mouth being fetid. The voice is thick, muffled, and nasal, and if the Eustachian tube be pressed upon, deafness and noises in the ears result. If the exudation be absorbed the symptoms speedily disappear. More commonly, fluxation may be made out in the tonsil in from two to four days, and rupture takes place some time between the fifth and tenth day. If this does not occur, in extreme cases suffocation may take place.

An abscess which points toward the soft palate often breaks sooner than one which shows a tendency to extend into the posterior wall of the pharynx. The amount of pus discharged is usually small.

Diagnosis.—The diagnosis may be impossible for a day or two. After a few days fluxation may be noticed in the tonsil.

Prognosis.—The prognosis may be considered as favorable to life. Complications do not occur if early incision into the tonsil is made.

Treatment.—Salines and cathartics are useful. Early incision of the affected tonsil should be made. The salicylates in some cases showing marked rheumatic tendency sometimes relieve pain. Local applications of ice or poultices are a relief to some patients. Cocain is of little benefit. For the severe pain, opium in some form is necessary. When fluxation can be elicited, free incision should be practiced.

CHRONIC TONSILLITIS.

Synonyms.—Mouth breathing; chronic nasopharyngeal obstruction.

Etiology.—The disease appears most frequently in children and young adults. It may follow some of the eruptive fevers, especially diphtheria. It is sometimes congenital.

Pathology.—It may be unilateral or bilateral, and consists of hyperplasia of the lymphoid structure of the tonsil. The interstitial stroma in some instances is increased, and the

organs then become firmer in consistency. Adenoid vegetations are sometimes present. Catarrh of the nasal pharynx usually accompanies this condition.

As a result of long-continued mouth breathing deformities of the chest arise. These deformities may be one of three varieties:

(*a*) Pigeon-shaped breast, or chicken-breast, in which form the prominence of the sternum and lateral depressions known as "Harrison's grooves" occur (see p. 53).

(*b*) The barrel-shaped, or inspiratory chest (see p. 51).

(*c*) The funnel-shaped chest (Trichterbrust; see p. 53).

Symptoms.—Breathing at night is laborious. The child has attacks of night-terrors. Snoring is common; the throat becomes dry, and cough occurs. Stuttering and habit chorea develop in some instances. The voice is altered, often becoming husky and having a decided nasal twang. Swallowing is difficult. The patient often has a vague, listless expression. Children with enlarged tonsils are liable to "colds" and attacks of acute tonsillitis. They are also often subject to attacks of croup. Upon inspection of the mouth the enlarged tonsil can readily be seen.

Treatment.—The treatment consists in removal of the hypertrophied tonsil. This may be done either by the tonsillotome or, preferably, by the cautery. Local applications of iodin and chromic acid are useful in shrinking the tonsil.

DISEASES OF THE PHARYNX.

ACUTE PHARYNGITIS.

Definition.—Acute inflammation of the pharynx. This occurs in association with inflammation of the soft palate and the tonsils.

Synonym.—Sore throat.

Etiology.—Exposure to cold, bad air, etc., may be etiologic factors. There is frequently a connection between the diseases of the stomach and diseases of the pharynx. Smoking, use of alcohol, and highly seasoned food may be predisposing causes. Nasal obstruction is an important cause. Occasionally the disease is epidemic. It may be of rheumatic origin.

Symptoms.—Constitutional symptoms are rarely marked. There may be a subfebrile temperature. There is a scratchy

feeling in the throat, with a frequent desire to swallow. The tickling in the throat may give rise to a dry cough. Upon examination the pharynx is at first dry and reddened; later a grayish-white secretion may be seen. The attack lasts from two to six days.

Treatment.—Inhalations of steam are grateful. Hot gargles are of decided use, hot milk being perhaps best for this purpose. A mild purge may be given at the onset.

CHRONIC PHARYNGITIS.

Synonyms.—Chronic follicular pharyngitis; chronic granular pharyngitis; and, when the dry variety occurs, or the atrophic form, it is known as pharyngitis sicca.

Etiology.—The disease begins in childhood, but its manifestations are often masked by hypertrophied tonsils. When the condition persists in adult life it causes much annoyance. The excessive use of the voice is frequently a causative factor. Pharyngitis sicca occurs from previous or associated atrophy of the turbinated bones.

Symptoms.—Dull pain in the throat upon speaking or swallowing, hoarseness, dry cough, and tickling in the throat are common symptoms. Examination will show bright red masses on the pharynx, the whole pharynx rarely being of the same color. The uvula is not necessarily elongated.

Treatment.—The hypertrophied follicles may be removed by galvanocautery. In the dry form the nasal pharynx should be frequently cleaned, sprays of menthol and benzoated glycerin being useful.

RETROPHARYNGEAL ABSCESS.

This is most frequent in children. It may result from caries of the cervical vertebra, and is sometimes a sequel of the infectious diseases, particularly diphtheria and scarlet fever.

Symptoms.—The patient becomes restless, and swallowing is difficult. There are changes in the voice. Pain is a constant symptom. On inspection a projecting mass may be noted upon the posterior wall of the pharynx. Fluctuation may be noticed upon palpation.

Treatment.—The treatment is surgical.

DISEASES OF THE ESOPHAGUS.

ACUTE ESOPHAGITIS.

Definition.—Acute inflammation of the esophagus.

Etiology.—This is a rare affection. It may result from exposure to cold. Hot and very cold drinks, alcohol, tobacco, and irritating substances, such as mustard, may cause it. Drugs, especially if irritating and remaining for some time in the esophagus, may cause the condition, and may lead to spasmodic contraction. The disease may arise from extension due to gastric or pharyngeal affections. It occurs in some of the infectious diseases, such as diphtheria and variola. The commonest cause is from the swallowing of corrosive substances; other causes are injuries from foreign bodies, spinal caries, abscess of the mediastinum, and suppurating tracheal and bronchial nodes.

Pathology.—The amount of inflammation depends upon the character of the irritant. It may be of the simple catarrhal variety, or sometimes of the ulcerative form, often leading to extensive formation of cicatricial tissue and contraction. It is rarely phlegmonous or pseudomembranous, which usually results from extension. From the lodgment of a foreign body acute suppurative inflammation may follow, and this may terminate in gangrene. Hemorrhage may take place from acute inflammation.

Symptoms.—The principal symptom is difficulty in deglutition, which may last for days or weeks. Attempts at swallowing are accompanied by burning, which may continue for several hours after partaking of food.

Regurgitation of food may occur before the food reaches the stomach. From the small amount of food taken the nutrition suffers, and rapid wasting may be a symptom.

In the purulent form, fever with slight rigors, and even with pronounced chills, may be present.

Blood, pus, mucus, and shreds of necrotic tissue may be brought up if the process be suppurative or gangrenous. Thirst is often pronounced. It is often difficult to differentiate between the mild and severe forms of the disease.

The esophagoscope may reveal the extent of the inflammatory process.

Prognosis.—In the simple catarrhal form the prognosis is

favorable. In the suppurative varieties death may result from perforation, gangrene, or subsequent stricture.

Treatment.—Fluid diet should be given until the acute symptoms subside. If liquids can not be taken, nutritive enemata may be substituted. Cracked ice is often grateful to the patient, and soda bicarbonate, placed upon the tongue and slowly swallowed, often gives relief. The salts of bismuth in emulsion, or the dry bismuth salts, placed upon the tongue and swallowed without water, may lessen the pain. Hydrochlorate of cocain in half-grain doses may be beneficial. It may become necessary to administer opium to relieve the suffering of the patient.

CHRONIC ESOPHAGITIS.

Etiology.—This is produced by repeated attacks of the acute or the subacute form. All causes which give rise to the acute variety give rise to the chronic.

Passive congestion of the esophagus, such as may occur from pulmonary or cardiac diseases, may cause the affection, and it frequently accompanies tubercular and syphilitic lesions.

Pathology.—Chronic esophagitis may be either of the chronic catarrhal variety or of the atrophic form, in which there is new-formed connective tissue present, showing a tendency to contract, causing stricture.

Symptoms.—The disease may exist without giving rise to any symptoms. Occasionally there is deep substernal soreness, especially if the passage of food gives rise to difficulty and pain upon swallowing.

Diagnosis.—The diagnosis can not be made with accuracy, but must be made by exclusion. The esophagoscope may show the true nature of the affection.

Prognosis.—The prognosis depends upon the cause.

Treatment.—The treatment is the same as in the acute variety.

SPASM OF THE ESOPHAGUS.

Synonym.—Esophagismus.

Etiology.—This condition occurs frequently in hysteric patients and hypochondriacs. It takes place in affections such as chorea, epilepsy, and particularly hydrophobia. It may accompany the lodgment of foreign bodies in the esophagus.

The so-called idiopathic form occurs in females of nervous temperament. Occasionally it is found in elderly males.

Symptoms.—The patient complains of difficulty in deglutition, and in the most marked cases liquids may be regurgitated. The attack usually comes on suddenly, with or without substernal pain. The passage of the bougie may be temporarily arrested at the point of the spasm, which soon relaxes, with or without slight effort. The condition is rarely fatal.

Diagnosis.—The diagnosis, as a rule, is not difficult. The occurrence of the disease in young persons of neurotic temperament, and the ease with which the esophageal bougie passes, are diagnostic. In elderly persons malignant stricture must be excluded.

Treatment.—The passage of the bougie often gives prompt relief. Tonics are necessary, and the general health should be looked after.

STRICTURE OF THE ESOPHAGUS.

Etiology.—This may be due to congenital narrowing; healed ulcers, resulting from corrosive poison and syphilis; the growth of tumors in the walls of the esophagus; external pressure by aneurysms, enlarged lymphatic glands, tumors, and rarely from pericardial effusion.

Pathology.—The stricture may occur in any part of the esophagus, and under rare circumstances involves nearly the whole tube. In the greater number of cases it is found either high up near the pharynx or low down near the cardiac extremity of the stomach. The stenosis may be either extreme or slight.

Symptoms.—When the stricture appears low down, the esophagus is commonly dilated and the walls hypertrophied. When it is high, food is generally rejected at once, whereas if the stricture be low down a considerable quantity may collect before regurgitation. The rejected material is alkaline in reaction; this and auscultation showing that it has not reached the stomach.

The constitutional symptoms depend greatly upon the cause, whether it be a malignant growth or a stricture resulting from a cicatrix.

Emaciation and marked anemia soon become pronounced if the stricture be complete.

Prognosis.—Prognosis depends upon the nature of the stricture, whether it be simple or malignant. In simple stricture resulting from cicatrix frequent dilatation renders the prognosis favorable.

Treatment.—Gradual dilatation by the esophageal bougie may be practised; however, great care should be exercised in its passage, especially if malignant disease with ulceration be suspected. In this event it is safer to use a soft, flexible tube. Rectal alimentation is frequently necessary to sustain life. Surgical interference is often of great value. Gastrotomy may be performed.

TUMORS OF THE ESOPHAGUS.

These are usually malignant, carcinoma being the most frequent. It may be primary or secondary. It is more prevalent in males than in females. Its most common seat is in the upper third of the esophagus, but there is much difference of opinion as regards the most frequent location.

The most common form of carcinoma found is the squamous cell variety; however, scirrhous and encephaloid cancers have been noted. Suppuration frequently results. Colloid degeneration of these tumors has been observed. Secondary deposits are most frequently found in the surrounding lymph-glands, sometimes in the lungs, liver, kidneys, and other organs.

The growth may involve the entire circumference of the esophagus, or may be only partial. Perforation and hemorrhage sometimes occurs as a result of ulceration.

Sarcoma of the esophagus is rare.

Of the benign epithelial tumors, papillomata are the most frequent. Of the benign connective-tissue tumors, fibromata and myomata have been found. If the tumor causes stricture, dilatation of the upper part of the esophagus frequently follows.

Symptoms of Carcinoma.—Difficulty in swallowing, first of solids and later of liquids, coming on gradually, is suggestive of this condition. There is pain upon swallowing, made worse by forcible attempts at deglutition. Occasionally painful deglutition may come on suddenly. The pain may be felt between the shoulders. If the growth appears in the lower part of the esophagus the pain in swallowing is not so marked.

Regurgitation of food is frequent, the vomited matter often containing blood-streaked mucus, sometimes even sloughs of the growth. The dysphagia is progressive. Hunger is a pressing symptom, the breath is offensive, and hiccup, associated with thirst and dryness of the mouth, is present. Rapid emaciation, with marked loss of weight and great debility, are pronounced features.

The cachexia of malignant disease shows itself in well-marked cases. The leukocytosis of malignant diseases is sometimes absent, for in starvation the leukocytes tend to decrease.

Symptoms of Sarcoma.—Sarcoma of the esophagus generally occurs in younger individuals. The condition is extremely rare as a primary growth. The symptoms are those of rapid emaciation and those just enumerated.

Benign Tumors.—The benign tumors simply cause symptoms of obstruction, but may produce death.

Course and Progress of Malignant Disease.—The course of the disease is rapid, death being due either to exhaustion, starvation, sepsis, pulmonary, or other complications.

Prognosis.—The prognosis is grave.

Treatment.—The treatment is usually surgical. Gastrotomy may prolong life. Opium should be given to relieve pain. Rectal alimentation may be employed.

DILATATION OF THE ESOPHAGUS.

This may be a simple cylindric dilatation as the result of obstruction, or the formation of a diverticulum. The latter is usually produced by traction from without, or may also be caused by internal pressure, and arises most often at the junction of the esophagus and the pharynx. It is more often found in males than females, and is most prevalent after the fortieth year of life.

RUPTURE OF THE ESOPHAGUS.

This may result from severe vomiting, especially if the wall of the esophagus has been previously diseased.

DISEASES OF THE STOMACH.

GASTRITIS.

(*a*) Simple acute gastritis; (*b*) severe acute and toxic gastritis; (*c*) phlegmonous gastritis; (*d*) chronic gastritis.

SIMPLE ACUTE GASTRITIS.

Synonyms.—Acute catarrh of the stomach; acute catarrhal gastritis.

Etiology.—Simple gastritis is a common affection. The disease may be primary or secondary. In the majority of instances the condition is due to irritants, either thermic or chemic, that come in contact with the mucous membrane of the stomach, producing an acute inflammation. Food either too hot or too cold, spices, drugs, and poisons, may have this influence upon the mucous membrane. Large quantities of food remaining in the stomach for some time may give rise to an acute atony, and produce gastritis. Decomposed food, especially such as is taken in the warm seasons, may cause acute gastritis, producing a form of *ptomain poisoning*.

It may be due to a fungous growth in the stomach, which is spoken of by some writers as *mycotic gastritis*. Parasites, such as the larvæ of some flies, and ascarides sometimes may be etiologic factors. Alcohol is probably the most common cause. The disease which gives rise to catarrhal gastritis is particularly acute parenchymatous nephritis, as was first pointed out by Fenwick. The disease also occurs in the course of scarlet fever, erysipelas, measles, and variola.

Pathology.—The surface of the stomach is usually covered with a thick, ropy, mucous or mucopurulent exudate. It may be streaked with blood. The epithelial lining shows granular degeneration and desquamation, with some infiltration of leukocytes in the submucous layer; less commonly there is hemorrhage.

Symptoms.—The disease may show all grades of severity. As a rule, fever is not present. The appetite is lost. There is a sense of pressure and weight in the epigastric region, with nausea and eructations, these symptoms being followed by vomiting of the foul, sour, scarcely digested remains of food. More or less mucus is mixed with the vomited material, which is acid in reaction, and sometimes contains bile.

The tongue is covered with a thick, grayish coating. It is swollen, and the margin shows the indentation of the teeth. The patient usually complains of a sour, disagreeable taste in the mouth, and shows great aversion to food. Thirst is increased. The epigastric region appears swollen, and is tympanitic and painful. Headache, with some vertigo and lassitude, is usually present. The urine is decreased in amount, high-colored, and contains uric acid, and often indican.

The pulse is rapid, small, and compressible. In the mild cases recovery soon follows. In the severer forms vomiting becomes frequent, the patient continuing to suffer from the symptoms enumerated. The condition may go on and produce a similar disturbance in the bowel. Constipation is at first the rule. Should the duodenum become affected, jaundice may make its appearance.

Occasionally, herpes labialis appears in those cases in which fever is present. It is of a marked remittent character; however, the normal temperature is soon reached and maintained.

Prognosis.—The prognosis is favorable; relapses, however, are common.

Treatment.—Rest is important, many cases recovering without the use of drugs. Calomel and castor oil, especially in children, should be administered. The diet should be restricted for a time. In severe cases it may be necessary to promote vomiting by the use of an emetic or the administration of warm water. In some cases lavage is indicated. Small pellets of ice should be given to relieve thirst.

SEVERE ACUTE AND TOXIC GASTRITIS.

Etiology.—Many authors prefer the term toxic gastritis, and probably most of the severe forms are due to toxic influences. The poisons which directly influence the mucous membrane of the stomach, especially in concentrated solutions, are the mineral acids, carbolic acid, the caustic alkalies, alcohol, phosphorus, arsenic, corrosive sublimate, potassium chlorate, potassium cyanid, and others. These chemicals produce a severe form of gastritis. They give rise to symptoms in varying grades of severity, acting either upon the empty or partially filled organ.

Pathology.—The inflammation excited by the causes mentioned in the etiology is of an acute variety. Large areas of

necrosis, and sometimes large ulcers, are formed when the necrotic tissue separates. Hemorrhage frequently results. Microscopically, leukocytic infiltration, areas of hemorrhages, dilatation of the blood-vessels, and necrotic tissue will be noted in the submucous, and sometimes muscular and peritoneal coats.

With healing, the extensive cicatrix causes great deformity, which may produce pyloric or cardiac stricture. Leukocytosis may be found.

Symptoms.—Promptly upon the ingestion of toxic substances the symptoms of a general intoxication develop. If the poison be taken in fluid form, the mouth, pharynx, and esophagus are likely to be first affected. Pain upon deglutition, felt in the pharynx and esophagus, under the sternum and epigastrium, quickly appears. It is severe in character, often described as burning. This is soon followed by vomiting, which is frequently repeated, accompanied by much nausea. The vomited matter contains particles of food, mixed with mucus and blood, and sometimes shreds of the mucous membrane of the stomach. Upon examination of the epigastric region decided tympany may be noticed, although this is not by any means constant; occasionally retraction in this region may be found.

Palpation of the epigastric region causes pain. The face is pallid, covered with cold sweat, and the expression shows evidence of suffering. The pulse rate is increased in frequency, the pulse being small and compressible. The extremities are cold and cyanotic. The respiration is increased, thoracic, and superficial. In aggravated cases peritonitis and perforation may occur. Death is due to collapse in many persons. These acute and serious symptoms only develop from corrosive acids or alkalies, when death may occur in from an hour to a day or two.

When recovery ensues, stenosis of the esophagus or pylorus may form, or the disease may merge into the chronic variety.

Diagnosis.—The history of the case is important. The evidence of inflammation upon the lips, tongue, or pharynx may show the nature of the poison swallowed. The acute onset and the symptoms just described render the diagnosis easy.

Prognosis.—The prognosis depends upon the poison and the amount swallowed. Toxic gastritis is always a very serious affection.

Treatment.—The indication is to get rid of the poison promptly, and to administer the proper antidote as soon as possible. Should the toxic agent not give rise to vomiting, the stomach must be washed out at once. Emetics which act promptly should be employed. Diluent drinks, frequently repeated, are of value. Purges act too slowly, hence their use is not indicated. Heart tonics must be used if signs of failing circulation occur. Opium in some form is often necessary to relieve pain.

PHLEGMONOUS GASTRITIS.

Etiology.—The disease is more prevalent in men than in women, in early adult or middle life. Alcohol is an important etiologic factor, probably being the predisposing cause in one-fifth of all cases. The disease may immediately follow the partaking of a heavy meal, after excessive eating, or periods of fasting. Injury may give rise to the condition. Among the primary factors the so-called idiopathic form of blood-poisoning, or septic infection, is the principal cause. Occasionally the condition may be secondary following injury or operation, such as from ulcers or cancers.

Pathology.—The phlegmonous inflammation may be localized or diffused. When localized, the abscess may be quite large, and rupture into the cavity of the stomach.

On examination of the stomach-wall it will be found greatly thickened. Ulceration may be present, involving the submucous coat. Microscopically, a dense leukocytic infiltration will be noted, as well as necrotic areas. Micro-organisms are found in great numbers, most often the streptococci. The muscular and serous coats may be involved. Pyemic manifestations may be noted in other organs.

Symptoms.—The symptoms are not always characteristic. The onset is sudden. The epigastric pain and vomiting are most constant. The disease has been divided into two periods, the symptoms before and after peritonitis has set in. Pain is present in the majority of cases, which is often violent, cutting, or burning in character, and commonly appears upon the first day of the disease. It increases in severity, and rarely subsides for any length of time. Often it is localized to the region of the epigastrium, but becomes general with the onset of peritonitis. The increased signs of resistance upon palpation are noticed in the epigastrium at an early period, these becoming most marked when peri-

tonitis occurs, the abdomen then becoming prominent, swollen, and tympanitic. Vomiting is an almost constant symptom. It may be preceded by anorexia and nausea, and often occurs upon the first day, sometimes being kept up continuously, and only ceasing with death. The vomited matter is at first watery, containing food and mucus, but it soon becomes tinged with bile, being yellow or greenish in color.

According to Reigel, pus has never been found in the vomited material. A slight degree of jaundice appears. Fever is usually present, the temperature ranging from 100° F. to 104° F. The pulse is full, strong, and but slightly accelerated; later it becomes rapid, feebler, and irregular. There may be diarrhea or constipation; occasionally they alternate. The tongue is dry and covered with a white coat. Thirst and anorexia are prominent. Wandering delirium, followed by coma and collapse; and the "typhoid state" sets in shortly before death.

Diagnosis.—The diagnosis depends upon the sudden onset, the localized pain, the bilious vomiting, the severity of the symptoms, and the accompanying peritonitis. The diagnosis must often be made by exclusion.

Prognosis.—The prognosis is hopeless. The course of the disease is rapid, and the duration brief.

Treatment.—The treatment is purely symptomatic. Rectal alimentation must sometimes be resorted to.

CHRONIC GASTRITIS.

Synonyms.—Chronic gastric catarrh; chronic dyspepsia.

Etiology.—The conditions giving rise to acute gastritis cause the chronic form. Free indulgence in acid substances may give rise to the condition. Alcohol in a concentrated form may act as an irritant. Overeating, especially of rich foods that produce fermentation, is a causative factor; also rapid eating, and ingestion of great quantities of food with ice-water at meals, as is so habitual with Americans, are important causes.

Carcinomata of the stomach and gastric ulcers frequently are accompanied by gastric catarrh. Bright's disease and pernicious anemia are frequent causes. Obstruction of the return flow of blood from the stomach, such as results in atrophic cirrhosis of the liver, valvular disease of the heart, and pulmonary diseases, may bring about the condition.

Pathology.—The stomach is usually dilated, although

sometimes it is decreased in size. The mucous surface is covered with a thick layer of mucus ; sometimes the membrane is discolored. The veins may be distended and tortuous.

On microscopic examination the epithelium and glandular structures are found almost constantly atrophied. Connective tissue is increased, sometimes causing increased thickness of the stomach-wall ; this is especially true when the stomach is decreased in size.

Symptoms.—The disease is chronic in its nature, and persists for a variable period. The early symptoms are a sensation of fullness after eating, sometimes accompanied by vomiting, eructation of sour gases, and palpitation of the heart. The tongue is coated with a grayish-white fur, and is moist. There may be pain after the ingestion of foods ; most frequently, however, there is pain at all times upon pressure in the epigastric region, which will be very tender. Loss of weight and marked secondary anemia usually accompany the condition. In some instances the appetite becomes variable, and there is craving for certain kinds of food. The bowels are usually constipated, in many cases obstinately. Cough, sometimes spoken of as stomach cough, accompanies the disease. Nervous manifestations, such as headache, depression of spirits, melancholia, and irritability, not infrequently appear as manifestations.

Gastric Contents.—Examination of the gastric contents after the administration of a test-meal reveals the following : Large quantities of mucus are present. Hydrochloric acid is most commonly diminished, and rarely completely absent, but sometimes the hydrochloric acid is found normal. Lactic acid may occasionally be present, especially when the material for examination contains large quantities of mucus.

Diagnosis.—The diagnosis of chronic gastritis is often difficult, and depends upon the gradual onset, distress after eating, and examination of the stomach-contents. The diagnosis between malignant diseases of the stomach and gastritis is often extremely difficult.

Treatment.—The treatment consists in the careful regulation of diet. Food should be masticated thoroughly and taken in small amounts. Fats, alcohol, and sugar should be avoided as much as possible. In severe cases a liquid diet, milk preferably, should be instituted. Medicinal treatment consists in the administration of hydrochloric acid well diluted, before meals. Pepsin, pancreatin, and bitter tonics have been

found of use. Constipation should be treated by such laxatives as cascara sagrada, and occasionally a mild calomel purge or salines are beneficial. Systematic lavage is of great use in the majority of cases; electricity and massage of the abdomen may be found of service.

DYSPEPSIA.

This is a term used to designate imperfect, difficult, or painful digestion.

Etiology.—The condition is due to many causes. Irregular meals, improper food, swallowing food without thorough mastication, cold and hot drinks, and the abuse of alcoholic liquors are all causative factors.

Nervous influences play an important part in the causation, such as mental overwork immediately after meals, bad news, worry, anxiety, and dissipation.

Want of exercise is important. The influences of microbes, especially those in the mouth, may give rise to forms of dyspepsia.

Finally, the disease is symptomatic in many affections and constitutional diseases. It occurs in adhesions of the intestines, in cases of floating kidney, and is often one of the first symptoms of incipient phthisis.

Dyspepsia is very apt to lead to chronic gastritis, and often is associated with neurosis of the stomach.

Symptoms.—The symptoms are extremely variable. Commonly there is coated tongue, anorexia, and even disgust for food. Nausea and vomiting may be important symptoms. There may be a feeling of depression in the epigastrium, pain, fullness, eructation, pyrosis, acidity, constipation, and diarrhea.

Any one of these symptoms may assume special prominence and the others be masked. Fever is rarely present. The urine may contain excess of solids, but otherwise is usually normal.

In forms of dyspepsia characterized by great flatulence, accelerated and disordered action of the heart from pressure of the overdistended colon upon the precordial spaces may take place.

The patients are often sleepy after meals, and unfit for mental or bodily exertion. In the so-called "nervous dyspepsia" there may be headache, vertigo, anesthesia, hyperesthesia, paresthesia, and occasionally even delusions and hallucinations may take place.

Diagnosis.—It is always necessary to exclude organic diseases of the stomach and other constitutional affections, especially if these symptoms should occur in the aged. The occurrence of these symptoms, with the history of want of exercise, hasty eating, improper food, etc., will usually give the clue to the true nature of the illness. It must not be forgotten that this disease also occurs in childhood.

Prognosis.—In simple, uncomplicated cases the prognosis is favorable.

Treatment.—It is important to instruct the patient to thoroughly masticate the food, and a restricted diet should be instituted. Large quantities of fluids had better not be allowed at meals. Oils, starches, and sugars should as far as possible be excluded from the daily dietary. Systematic exercise, in which fatigue should be avoided, should be insisted upon.

Abdominal massage is often of decided value, especially if constipation be a symptom. The drugs which have been found to be especially useful are the mineral acids, the alkalies, bitter tonics, and digestive ferments, such as pepsin and pancreatin. The constipation may be relieved by suitable drugs.

ULCER OF THE STOMACH.

Definition.—A disease of the stomach, characterized by local pain, vomiting, and hematemesis.

Synonyms.—Perforating ulcer; round ulcer; simple ulcer; peptic ulcer.

Etiology.—The disease is common, especially in the female sex, occurring about twice as often in females as in males, most often between the ages of twenty and forty. The disease is frequent among the poorer classes. It is often associated with other ailments, especially the various forms of anemia, tuberculosis, syphilis, scurvy, septicemia, and pyemia.

Etiologically, there is an undoubted connection between chlorosis and gastric ulcer. Trauma plays some part in the production of gastric ulcer, but this connection must be put down as rare. By most authorities it is claimed that heredity may have something to do with the production of the disease.

Pathology.—The most common situation of the ulcer is near the pyloric end of the stomach, on the posterior wall, near the lesser curvature.

The ulcers may be either single or multiple. They are usually circular in outline, varying in diameter from a few millimeters to six or eight centimeters, or may be oval; it is frequently funnel-shaped, but sometimes it is punched out, the edges being quite irregular and overhanging. The depth varies from a slight erosion to a deep ulcer with indurated, greatly thickened walls. Embolism and thrombosis may give rise to ulceration. In some instances the ulcer perforates, giving rise to either local or general peritonitis. When the inflammation is local, adhesions may spring up from surrounding structures and a subphrenic abscess be formed. Such abscesses are occasionally quite large, and have been known to rupture into the pleura, pericardial sac, liver, spleen, pancreas, or they may produce general peritonitis. If the ulcer be situated on the anterior wall, perforation will be followed by general peritonitis.

Copious hemorrhage results from the necrotic process, involving the vessels. The glandular elements of the stomach are more active, so that hydrochloric acid is increased.

When healing of the ulcer occurs, a stellate scar is often produced, which is not infrequently found during postmortem examination. From the contraction of the scar, if it be situated in the neighborhood of the pylorus, not infrequently stenosis of this orifice is produced; if near the center of the organ, the *hour-glass stomach* may result. If the ulcer be situated near the cardiac orifice, the healing process may cause a stricture there. If the ulcer is of long standing, carcinoma has been known to follow.

Symptoms.—In some cases the symptoms are absolutely characteristic, whereas in other cases it is almost impossible to diagnosticate the condition. The most constant of the symptoms is the pain, which occurs paroxysmally, and is localized. It commonly appears during digestion, and is aggravated by the taking of food. The most common situation of the pain is at the ensiform cartilage (pit of the stomach). The pain is sometimes described as circumscribed, gnawing, burning, and aching, and occasionally radiates to the back. The slightest pressure even of the clothing is sometimes unbearable. Occasionally the pain is entirely absent.

The quality of food is of great influence in the production of pain. Very hot and cold fluids do not generally produce pain, but solids are almost certain to give rise to a more or less well-defined paroxysm. Tenderness is common in the

entire epigastric region. A spot painful upon pressure may be found posteriorly in the dorsal region. The next symptom in frequency is vomiting. This generally takes place in from one to three hours after the ingestion of food. It may occasionally be due to the great pain. There are associated nausea, pyrosis, and eructation of gas of varying grades of severity. The vomited material consists of the stomach-contents, being markedly acid in reaction, due to the increase of free hydrochloric acid, often containing blood-streaked mucus. The blood is often bright red in color, exceedingly rarely of the "coffee-ground" variety. Microscopically, epithelial cells, red blood-corpuscles, and masses of granulation tissue are often observed.

Hemorrhage may be so copious as to lead to fatal collapse. Generally dyspnea is present. The tongue is clean, moist, and red, and is rarely coated. Thirst is increased and the appetite is variable. The patients have desire for food, but on account of the pain refrain from taking it. Occasionally there is increased appetite and even bulimia. It is rare for the appetite to be entirely wanting. There are pyrosis and cardialgia. Constipation is often extreme; rarely there is diarrhea.

The patient is almost certain sooner or later to show a high grade of anemia of the chlorotic type, but sometimes the erythrocytes are greatly reduced in numbers. After profuse hemorrhage numbers of nucleated red blood-cells may be observed with the accompanying leukocytosis. Digestive leukocytosis is said to be increased in gastric ulcer. Dysmenorrhea and amenorrhea are common. Fever is not a symptom of gastric ulcer. Loss of weight is not marked, excepting where stenosis of the pylorus or cardia should occur.

Complications.—Perforation, with general or local peritonitis, carcinoma, and occasionally pernicious anemia are important complications. Subphrenic abscess, as described in the pathology, results from perforation, circumscribed peritonitis following. The abscess is situated beneath the diaphragm, more frequently to the right than to the left of the median line. If situated on the left side, increased area of dullness will be noted in the left hypochondriac region, and the heart and lungs may be displaced upward.

Great tenderness upon pressure is noticed in the region corresponding to the abscess. The symptoms are not always diagnostic. The temperature is usually of the septic type.

Hiccup, due to irritation of the diaphragm, may be present, and there may be edema of the lower parts of the thorax posteriorly.

Subphrenic abscess may be due to other causes besides perforating gastric ulcer. Ulcer of the duodenum, perforation of the appendix, and abscess of the liver may also give rise to it; however, the majority of cases are due to ulcer of the stomach.

It is especially important to diagnosticate an abscess which occupies the part beneath the diaphragm. The sign that the lung dilates upon inspiration, that pure vesicular breathing is present, are important points in this connection. The lower parts of the thorax show bulging of the interspaces; edema in these parts may be present. The heart is but slightly or not at all displaced, but the liver is usually found low down in the abdomen, at or below the umbilicus.

Subphrenic abscesses may be so small and occupy so limited an extent that the physical signs may be entirely absent.

Clinical Varieties of Ulcer of the Stomach.—The Hemorrhagic Variety.—This may have either an acute or a chronic course. In either case, the variety is characterized by copious and severe hematemesis. This may be so excessive that the patient dies as a result of the hemorrhage. Occasionally the disease runs an acute course, and after severe blood loss rapid recovery follows. In this form the stools are very apt to be tarry and show the presence of blood.

The Acute Perforating Variety.—The symptoms of the ulcer may be attended with slight or no dyspeptic symptoms. Suddenly, and without apparent cause, perforation develops, which in the majority of cases rapidly leads to death. In this variety the ulcer is almost always situated upon the anterior wall of the stomach.

The Chronic Dyspeptic Variety.—In this variety the dyspeptic symptoms are most marked. The symptoms are those of a chronic gastric catarrh. There is pain, particularly after taking food, tenderness upon pressure in the epigastric region, and occasionally vomiting. There is marked hyperacidity, due to excess of free hydrochloric acid. This latter fact is important from a diagonistic standpoint.

The Gastralgic or Nervous Variety.—In this variety pain and tenderness assume prominence, the other symptoms being more or less masked.

The Variety Characterized by Vomiting.—This was first described by Lebert. It is characterized by almost continuous painful vomiting, the patient retaining little or nothing upon the stomach. Rapid wasting assumes alarming proportions in this variety.

The Cachectic Variety.—In this form the patient shows a high grade of cachexia. He is pale, and loss of flesh is marked, and the symptoms resemble carcinoma. These symptoms occur particularly in the later stages of long-standing ulcer appearing in elderly people. Hypersecretion is present, and cicatrices from old healed ulcers are apt to be noticed at the autopsy.

Course of the Disease.—The course of the disease is most often chronic, the condition having been designated *chronic ulcer* of the stomach. Even after apparent cure, relapses may take place from time to time, with a return of the initial symptoms. Sometimes in the aggravated form, as has already been indicated, some cases may run a more or less acute course.

Lebert has estimated the average duration of the disease as from three to five years. It may last longer than this, some cases having been observed that have run a course of from twenty to thirty years. The cases of long duration are most often complicated, particularly by stenosis of the cardia and pylorus.

Diagnosis.—In the diagnosis of ulcer it is not only important to diagnosticate the actual existence of the affection, but its position, and if possible the question of whether it be single or multiple, or whether it be complicated by some other affection.

The general diagnosis depends upon pain in the epigastrium, which is localized and paroxysmal, being increased by taking food, and the excess of free hydrochloric acid in the gastric contents. In connection with these symptoms, eructation, cardialgia, and vomiting make the diagnosis all the more certain.

Leube advises in all cases of doubt to make a therapeutic diagnosis—that is to say, all patients that are suspected of having gastric ulcer should be put upon treatment.

The differential diagnosis between ulcer and cancer of the stomach will be given in the description of cancer. The differential diagnosis between ulcer of the stomach and ulcer of the duodenum can only be made with the greatest difficulty. In ulcer of the duodenum the pain is felt over toward the

right parasternal line. The blood is not apt to be vomited, but passed in the stools. The pain occurs later after taking food than in ulcer of the stomach. The localized area of pain in the region of the ensiform cartilage is absent. Hyperacidity of the gastric contents may be absent.

Ulcer of the duodenum may arise after extensive cutaneous burns. It occurs more frequently in the male than in the female sex, and especially in alcoholics. Vomiting may also be absent in ulcer of the duodenum. Occasionally, jaundice is present.

Ulcer must be differentiated from attacks of gastralgia, especially taking place in neurasthenic and nervous subjects. The points that favor gastralgia are the variability of the appetite, longing for certain kinds of food, the irregularity with which the pain occurs after the taking of food, pain even being diminished by pressure in the epigastric region, and the absence of hematemesis. Hyperesthesia is sometimes present in this affection, but never assumes the grade that appears in ulcer.

It is exceedingly difficult to make out the exact position of the ulcer. Signs that point to the ulcer having its position in the anterior wall are excessive tenderness and the presence of tumor. Pain in the back and great vomiting point to the posterior wall. Great gastrectasis points to the pylorus or duodenum as the seat of the ulcer.

Prognosis.—The sooner treatment is instituted the more favorable the prognosis. Relapses, however, are common even in cases that are most carefully and cautiously treated. The mortality of ulcer of the stomach has been estimated as from 8% to 10%. As has already been indicated, the position of the ulcer is most important from a prognostic standpoint, the cases in which the ulcer is situated upon the anterior wall being the most dangerous.

Treatment.—Rest in bed is most important even in the mildest cases. Food should be withheld from the stomach for some time, rectal alimentation being resorted to. If great thirst is a prominent feature, small pellets of ice placed upon the tongue from time to time may be useful. Milk is the best food; it may be given either hot or cold, according to the individual taste of the patient. External applications over the epigastrium, such as hot poultices frequently renewed, are of value. Occasionally, turpentine stupes relieve pain. A diet consisting of neutral ice cream (vanilla) is grateful to many patients.

Many drugs have been given to effect a cure, among the most useful being bismuth in large doses, twenty to thirty grains, three to four times daily, salol, oxid of silver, carbonate of soda, carbolic acid, and cocain.

If the pain becomes excessive, opium and sometimes cannabis indica may be resorted to.

The treatment should be continued for some time after the symptoms have subsided, great caution being enjoined in reference to subsequent diet. The constipation should be relieved by means of Carlsbad salts or other salines. Lavage must be used with great caution, and especially in cases characterized by marked tendency to hemorrhage.

MALIGNANT TUMORS OF THE STOMACH.

CARCINOMA.

Synonyms.—Carcinoma ventriculi; malignant disease of the stomach.

Etiology.—The stomach is a very common seat of cancer. By some authorities it is claimed that no other organ shows primary cancer as frequently as the stomach (Riegel).

It is more prevalent in the male sex than in the female. The majority of cases occur between the fortieth and seventieth years of life. Prior to this time cancer of the stomach is quite rare, although cases of congenital cancer have been reported, one instance being that of a child five weeks old. It is very rare in the tropics, being most common in the temperate and colder climates. Heredity seems to play some part in the production of the disease, although this is questioned by some authorities. Cancer sometimes follows ulcer of the stomach, and this is not so rare as has been supposed. There is no evidence that chronic indigestion leads to cancer of the stomach.

Pathology.—The most frequent seat of carcinoma of the stomach is at the pylorus. According to Lebert, it is found in 51% of the cases: the lesser curvature is involved in 16%, the cardiac orifice in 9%, the greater curvature in 4%, and diffuse infiltration in 6% of the cases.

A number of varieties have been found, such as the encephaloid carcinoma, scirrhus carcinoma, squamous cell carcinoma, adenocarcinoma, and colloid carcinoma.

The gross appearance of carcinoma of the stomach varies

greatly. The involvement may be limited or quite extensive. The surface, as a rule, shows marked ulceration, and sometimes "cauliflower projection." The color of the growth is usually grayish red: the consistency is either soft or hard, depending upon the variety present. In the scirrhous form a hard, indurated mass is observed, which if situated at the pyloric orifice may cause complete obstruction.

In the encephaloid variety the tumor is soft. In the colloid variety it is gelatinous.

Microscopically, epithelial cells will be found proliferating in the lymph-spaces, involving, as a rule, all the coats of the stomach. In some instances, true adenomatous growths will be found in some parts of the tumor, being called adenocarcinoma. As in all carcinomata, blood-vessels will not be found in the collections of epithelial cells. In the scirrhous variety a great amount of fibrous connective tissue is present between the groups of cells.

The superficial area frequently shows necrotic changes and leukocytic infiltration. The blood-vessels are usually eroded, from which hemorrhage results. If stenosis of the pyloric orifice is present, dilatation of the stomach will be noticed. In some instances this is very marked, the lower border of the stomach often reaching to the level of the umbilicus or below it, and the pylorus being found in the right iliac fossa. If stenosis be present at the cardiac orifice, the gastric walls reveal atrophy, and the organ is decreased in size.

Adhesions of the stomach may form with the liver, the anterior abdominal wall, the colon, and the pancreas.

Metastasis to the lymphatic glands is most common; the liver is next in order of frequency. The omentum, intestines, pancreas, lung, spleen, pleura, and other organs have been found involved. If the blood-vessels be implicated, the tumor may spread by the blood stream, and general carcinosis result.

Perforation of the stomach from carcinoma may occur, but this is rare. The involvement of the gastric tubules, with their destruction, produces a decrease or absence of hydrochloric acid. Anemia is present in all cases. The erythrocytes, as a rule, are slightly decreased; in some instances the number may be increased, due to blood concentration, and in a few cases a marked decrease may be present. Hemoglobin shows a marked decrease, so that the chlorotic type of anemia is usually present. Leukocytosis (called malignant leukocy-

tosis) generally appears, but when starvation results, for example, if a tumor be present at the cardiac orifice, the number of leukocytes may be normal.

Symptoms.—The disease begins insidiously, with extremely variable symptoms, or the symptoms may be so pronounced that the diagnosis of carcinoma of the stomach can not be doubted. On the other hand, as Osler ("Philadelphia Medical Journal," February, 1900) has recently pointed out, the symptoms may be so latent that carcinoma of the stomach is found only upon the postmortem table.

The onset in the majority of instances is insidious, affecting individuals between the ages of forty and sixty years. The patient gives the history of having been free from general dyspeptic symptoms until recently, when anorexia, a sensation of fullness in the epigastrium, with eructation or other signs of a mild gastritis, appeared. In rare instances the disease may begin suddenly, sometimes following influenza; these cases are, however, exceptional.

At the beginning of the disease *dyspeptic symptoms are of a mild grade, but yield very stubbornly or not at all to treatment.* As a rule, the appetite is lost, and there is often actual disgust for food. Pain of some degree of severity is always present, although it is not so severe nor so marked as in ulcer. The pain is not localized, but is diffused, often radiating anteriorly and posteriorly.

Vomiting is a frequent and important symptom. In the greater number of cases it is late in its onset, and is especially marked in those cases in which the seat of the lesion is at the pylorus. The vomiting is quite characteristic, occurring every day or every other day. The vomited material is usually copious in amount, containing undigested particles of food, having a sour, offensive smell, even containing particles of blood, and large quantities of mucus. In some cases of carcinoma vomiting may be absent altogether. The quantity of the vomited material varies greatly; it may be from one-half to two or more liters. When large quantities are vomited *gastrectasis* is present. Occasionally there is bile in the vomited material.

Microscopic examination of the vomit reveals many bacteria. The Oppler-Boas bacillus is most constantly found, and it is supposed to be responsible for the formation of lactic acid. Red blood-cells and leukocytes are usually found, as well as epithelial cells. In some instances irregular

groups of epithelial cells are present, these being parts of the cancer.

The vomited matter may consist almost entirely of "coffee-ground" material, which is altered blood.

After the administration of a test-meal, the material for examination in almost all instances shows the absence of free hydrochloric acid and the presence of lactic acid.

The bowels are constipated. This may be due to the fact that very little food reaches the intestines. Emaciation of some grade develops in all cases, which is often extreme, the patient rapidly losing many pounds in weight.

The cancerous cachexia usually develops early. The most important phenomena are those which relate to the local examination of the affected parts. The tongue is thickly coated, being covered by a copious, tough layer of mucus. The lymphatics of the neck are sometimes enlarged. The temperature is almost always subnormal unless inflammatory complications occur.

Late in the disease edema of the extremities is noticed; rarely does ascites develop, which may be due to pressure upon the portal vein. Coma is not uncommon, v. Jaksch being the first to call attention to this symptom complex in malignant diseases. The urine shows no constant characteristic changes. Albumin and casts are present in a number of cases, and indican is frequently found. Glycosuria, peptonuria, and acetonuria are sometimes present.

Physical Signs.—Inspection of the abdomen gives important results. In advanced cases the belly wall is relaxed, and the tumor may be noticed in the affected area. These signs are intensified by inflation of the stomach by means of Seidlitz powder or other methods. Gastroptosis and gastrectasis may be best noticed by this method.

Palpation is of great importance. This produces tenderness, even upon slight pressure. To determine the exact position of the tumor, it is advisable to fill the stomach so that all the relations of the parts may be made out. In rare instances the tumor can not be felt. Tumors of the pylorus are movable toward the right and downward, rarely upward. Tumors of the posterior wall and of the lesser curvature can rarely be palpated. In advanced cases enlargement of the inguinal glands is noted, and nodular masses may be made out throughout the abdomen, indicating glandular involvement.

Complications.—A rare complication is tetany. It is especially observed in cases characterized by marked gastrectasis. Metastasis occurs in many organs.

Course of the Disease.—The duration of the disease varies. It may ordinarily be estimated as from one to two years, the hard carcinomata lasting longer than the soft varieties. Occasionally the duration of the disease is longer than two years.

Diagnosis.—The direct diagnosis depends upon the appearance of dyspeptic symptoms in a person between the fortieth and sixtieth years of life, with pain of varying grades of intensity, and tenderness upon pressure in the epigastric region, often increased by the taking of food, the vomited matter containing "coffee-ground" material, examination of the gastric contents by means of a test-meal, the absence of free HCl, the appearance of the tumor, the great and rapid emaciation, the cachexia, and the subnormal temperature.

Differential Diagnosis.—

Gastric Cancer.	*Gastric Ulcer.*
Most prevalent in the male sex, occurring between the fortieth and sixtieth years.	More prevalent in the female sex, in adolescence.
Marked emaciation, with cachexia.	Emaciation not present or slight; anemia marked.
Vomiting of "coffee-ground" material; rarely bright-red blood; vomiting late after taking food, often in large amounts.	Vomiting of bright-red blood common; rarely "coffee-ground" material; vomiting almost immediately after eating.
Absence of free hydrochloric acid; presence of lactic acid; pepsin diminished.	Excess of free hydrochloric acid.
Presence of palpable tumor.	No tumor.
Prognosis unfavorable.	Prognosis as to life is good.
Perforation and peritonitis rare.	Perforation and peritonitis more frequent.

Prognosis.—The prognosis is grave.

Treatment.—The most important point in the treatment relates to the early diagnosis of the condition, with the hope of operative interference. Medicinal methods are only palliative. They consist in the alleviation of the dyspeptic symptoms and the pain. To accomplish these ends, systematic lavage and the use of opium are the most satisfactory agents. Strength may be maintained by rectal alimentation.

SARCOMA OF THE STOMACH.

Sarcomata may be primary or secondary. They are most common in young individuals between the ages of twenty and thirty-five years. The symptomatology is not characteristic.

NONMALIGNANT TUMORS OF THE STOMACH.

Fibroma, fibromyoma, adenoma, papilloma, and lipoma of the stomach have been observed.

FOREIGN BODIES IN THE STOMACH.

Foreign bodies in the stomach are sometimes mistaken for tumors. The insane and hysteric women have been known to swallow hair, which may aggregate into a large mass. Many other bodies may be swallowed, such as false teeth, etc. Operative interference is necessary in some instances.

HEMORRHAGE FROM THE STOMACH.

Synonym.—Hematemesis or gastrorrhagia.

Etiology.—Hemorrhage from the stomach may arise from a number of conditions, such as cancer, ulcer, from local destruction such as occurs from acids or alkalies, active or passive congestion, especially from obstruction to the portal circulation, in the course of atrophic cirrhosis of the liver, and from mechanical injuries, such as wounds inflicted either internally or externally. It may also arise from constitutional disturbances, such as hemophilia, melena, pernicious anemia, and purpura hemorrhagica, also yellow fever, smallpox, and acute yellow atrophy of the liver. It has been known to arise in hysteria. The rupture of aneurysms may produce hemorrhage, and in some instances blood gains entrance to the stomach from other parts, most frequently being swallowed.

Symptoms.—If the blood loss be copious, there is faintness followed by syncope, coldness of the surface and extremities, sweating, and subnormal temperature, sighing respiration, small, weak pulse, occasionally accompanied by convulsive movement, and later blood may show itself in the stools, indicated by their tarry character.

Treatment.—The treatment is the same as in hemorrhage taking place in other internal conditions.

DILATATION OF THE STOMACH, DISPLACEMENTS OF THE STOMACH, AND OTHER DEFORMITIES.

Dilatation of the stomach is called *gastrectasis.* The synonym for dropping of the stomach is *gastroptosis.* The stomach varies as to size in different individuals, the normal adult stomach holding about 1700 c.c.

GASTRECTASIS.

Etiology.—The dilatation may be acute or chronic. It may be due to obstruction of the pyloric end, such as congenital stenosis, the cicatrices resulting from gastric ulcer, and from tumors, the most common being carcinomata. Thickening of the pylorus occasionally arises without apparent cause. The pyloric orifice may be narrowed from external pressure, tumors of various kinds, and adhesions. Foreign bodies, such as hair balls, coins, etc., may obstruct the pyloric orifice and give rise to the condition. The obstruction may take place in the duodenum from the causes just enumerated.

Motor insufficiency of the stomach may give rise to dilatation. This results from overeating and the ingestion of large quantities of fluids, and is frequently met with in beer-drinkers. It appears in chlorosis, tuberculosis, diabetes, and other chronic or exhausting diseases. The stomach-wall may be diseased and give rise to gastrectasis, which is common in chronic catarrhal gastritis.

Chronic dilatation is principally a disease of adults in middle life. If it occurs in old persons it should give rise to suspicion of malignant disease.

Acute dilatation is frequently a disease of young persons, of children, and particularly of infants, as, for instance, the "pot belly" appearing in rickety children. The dilatation may be only temporary, and may take place without injury to the organ.

Pathology.—The degree of dilatation varies, in some instances being enormous, the lower border of the stomach reaching far below the level of the umbilicus. The capacity of the stomach is sometimes greatly increased; in the cases mentioned by Blumenthal the vomited material weighed sixteen pounds. The stomach-walls are usually thicker than normal. Catarrhal inflammation, as a rule, is present. The

stomach-wall may reveal other pathologic changes, depending upon the causation.

Symptoms.—The symptoms are, general feebleness, anemia, emaciation, thirst, scanty urine, sallow and hollow-eyed face, feeble flabby coated tongue, pyrosis, chilliness, cyanosis, subnormal temperature, and nervous manifestations. Pain and vomiting are always present and most definitely indicate the nature of the affection. The pain is particularly felt after taking food. If associated with lesions of the cardia or pylorus, it is almost constant. It is often immediately relieved by emptying the stomach, as from vomiting or by siphonage. Thirst and hunger are common symptoms. The urine is scanty and often deficient in chlorids. It is frequently alkaline, containing triple phosphates, and albumin being often present. Constipation of an obstinate kind is conspicuous, occasionally alternating with diarrhea. Emaciation is often marked. The nervous symptoms consist in depression of spirits, melancholy, sleeplessness, tinnitus, vertigo, and visual disturbances. Occasionally syncope, convulsions, and even tetany may occur, the patient dying in coma.

Physical Signs.—Inspection.—Emaciation is marked. Often the distended outline of the diseased organ can be seen. Peristaltic movements of the stomach may also be noted. Occasionally inspection is negative. The outline of the stomach may be demonstrated by means of the X-rays, as described by C. L. Leonard, of Philadelphia, a bismuth mixture being introduced into the stomach so that the shadow is produced.

Palpation.—Occasionally upon palpation with slight pressure over the epigastrium a splash is produced. This is due to the enormously dilated stomach being partially filled with fluid. If a tumor is present it may be detected, particularly at the pylorus.

Percussion and *auscultation* are of very little value. Upon auscultation, sometimes, by shaking the patient, the succussion splash may be heard.

The stomach-tube must frequently be introduced beyond the normal limit in order to siphon out the contents. The amount of liquid which the stomach contains will determine the extent of dilatation. The presence of free hydrochloric acid varies, depending upon the cause.

Prognosis.—The prognosis depends upon the underlying condition. The disease is obstinate, and most often incurable.

Treatment.—Lavage, or siphonage as it is sometimes called, is of benefit in the majority of cases, care being taken not to overload the stomach and not to allow liquids to remain. Abdominal massage is of use in many instances, but should be practised by an experienced person. Electricity has also been recommended. Drugs have been found unsatisfactory. Laxatives should be administered from time to time. Bitter tonics have been found of use in aiding digestion. The diet should consist largely of solids. In some instances operative interference in relieving stenosis is of decided value.

GASTROPTOSIS.

Definition.—Downward displacement of the stomach.

Etiology.—This may result from the causes just enumerated in gastrectasis, and sometimes accompanies the displacement of other organs. Glènard described the downward displacement of the viscera known as *visceroptosis*. It sometimes results from pressure upon the abdomen. Certain occupations, as those of tailors and shoemakers, are prone to produce this affection. Tight lacing is also an important factor. It is most frequently encountered in adult life, occurring more frequently in women than in men. Chest deformities (kyphosis) may give rise to the condition. Repeated pregnancies may cause relaxation of the abdominal wall and thus be causative elements. It is associated with neurasthenia. Traumatism, chronic diseases giving rise to emaciation, peritoneal adhesions making traction upon the stomach, and the removal of large abdominal tumors are sometimes etiologic factors.

Symptoms.—The disease may exist without giving rise to symptoms. When symptoms arise they are generally due to functional disturbances of the organ, gastric atony taking place. There is often diminished gastric secretion, with symptoms of nervous dyspepsia. Upon inflation, the stomach is noted to be of normal size, but displaced. Constipation and colicky pains in the abdomen are often important features.

Prognosis.—The prognosis greatly depends upon the etiologic factor.

Treatment.—The treatment consists in the removal of the cause, if possible, and in massage, electricity, proper food, bitter tonics, and remedial agents which improve the general constitution. Constipation should be relieved by suitable remedies.

OTHER DISPLACEMENTS.

The stomach may be displaced to the right, to the left, or upward, from the pressure of tumors, or abdominal enlargements. Deformities of the stomach are sometimes found, such as the "*hour-glass contraction.*" The organ may be vertical, and rarely the greater part of the stomach is on the right side in the condition known as *transposition of the viscera.*

NEUROSIS OF THE STOMACH.

This may consist in increase or diminution of sensibility, in the increase or diminution of contractility, or in the increase or diminution of secretions. The so-called *nervous dyspepsia* is an important form of neurosis.

GASTRALGIA.

The condition consists in severe boring, rumbling, painful contraction in the epigastric region, extending from the xiphoid cartilage and radiating to the back, accompanied by syncope and signs of collapse. The condition may appear suddenly, without apparent cause, or may be due to slight pressure in the epigastrium. It may be accompanied by the sensation of the globus hystericus, bulimia, frequent micturition, and vomiting. It disappears after having lasted but a few moments. The attacks occur with marked irregularity, occasionally several taking place in one day, upon alternate days, or not recurring for months. Gastralgia is important on account of the attack sometimes simulating other affections, particularly intercostal neuralgia, ulcer of the stomach, attacks of hepatic colic, nephralgia, and enteralgia, and the *gastric crisis* of locomotor ataxia. The diagnosis of gastralgia should never be made unless these diseases can be excluded.

BULIMIA.

This may occur in association with the sensation of an empty stomach. There is a constant desire for food without satisfying the appetite, the sensation being accompanied by headache, palpitation of the heart, and syncope.

NEUROSIS OF SECRETION.

Hyperchlorhydria or hyperacidity.

This condition is exceedingly common in ulcer of the stomach, but may occur as a neurosis.

The diagnosis depends upon the examination of the gastric contents. It may be produced by highly seasoned foods, in alcoholic intoxication, and sometimes from great anxiety.

Symptoms.—The symptoms consist in pressure and fullness in the epigastrium after meals, occasionally accompanied by pain, acid eructations, cardialgia, pyrosis, and sometimes bulimia. Vomiting is rare.

The condition should only be diagnosticated when ulcer of the stomach can be excluded.

PERISTALTIC UNREST OF THE STOMACH.

This condition was first described by Kussmaul.

The continuous and repeated contractions of the muscles of the stomach are most marked after meals, and also occur in the interval between meals. Belching and eructation may occur. This condition can often be seen by inflation. Occasionally, painful contractions of the cardia and pylorus take place.

DIMINISHED PERISTALSIS OF THE STOMACH, OR ATONY.

Pyloric relaxation is a rare form of neurosis, and when it occurs the undigested gastric contents enter the intestinal canal.

Relaxation of the cardiac orifice gives rise to eructation, and regurgitation of food.

Prognosis.—The prognosis is favorable if the underlying cause can be removed.

Treatment.—The treatment consists in removing the underlying cause if possible. Change of scene, a long ocean voyage, and tonic treatment are of value.

DISEASES OF THE INTESTINES.

INTESTINAL CATARRH.

ACUTE AND CHRONIC CATARRHAL ENTERITIS.

Definition.—Acute catarrhal enteritis is an acute inflammation of the intestinal mucous membrane.

The condition is occasionally local, due to the inflammatory process involving limited parts of the bowel.

Synonyms.—Acute inflammation of the intestines; acute intestinal catarrh.

Etiology.—The disease may be primary or secondary. It is primary when due to errors of diet. Coarse food imperfectly masticated produces an inflammation in the stomach (gastritis), followed by an inflammatory condition in the intestinal tract known as enteritis. This inflammatory condition, although acute, is almost always confined to the mucous membrane, constituting intestinal catarrh. Unripe fruits and badly cooked vegetables are often causes, especially during the hot seasons. Spoiled meat, fish, and milk and ice cream that have become tainted with parasitic fungi, frequently excite violent catarrhal enteritis. Beer and wines that have undergone unhealthy fermentation, and excessive drinks of cold water, especially bad, foul water mixed with sewage, give rise to the condition. The affection may be due to sudden chilling of the body, especially during a copious perspiration, as from a plunge into cold water while sweating is going on. Traumatism may play some part in the etiology. Falls, blows, and kicks of the abdominal wall may cause the disease. Foreign bodies may act in the same way. Large gallstones, fecal masses, long-continued constipation, parasites such as intestinal worms, various micro-organisms, chemic substances such as the alkalies, mineral acids, corrosive sublimate, arsenic, and so on, may be causative factors.

The specific micro-organisms play an important rôle, especially the comma bacillus of Koch. The extremes of age are particularly liable to acute inflammatory conditions of the bowels. It is not quite clear how nervous influences affect the intestinal mucous membranes, but the observation has been repeatedly made that emotion, fright, and anger cause acute catarrhal enteritis.

Among the secondary causes are inflammations of the contiguous parts—for instance, peritonitis causes intestinal catarrh. Eczema of the anus may extend into the rectum. Injury to the mucous membrane, as from bougies or other foreign bodies, may excite catarrhal inflammation. Compression of the intestines from the growth of tumors, circulatory disturbances, especially those relating to the portal circulation, also conditions which relate to the heart and lungs, may give rise to intestinal catarrh. The disease commonly occurs in association with pulmonary tuberculosis, renal disease, malarial cachexia, syphilis, Addison's disease, cancer, and profound anemia.

Pathology.—The inflammation may be limited to a part or

may involve the whole of the small intestine. When limited to the duodenum, it is called *duodenitis;* to the jejunum, *jejunitis;* to the ileum, *ileitis;* to the colon, *colitis;* and to the rectum, *proctitis.* In the greater number of cases the whole intestinal mucous membrane is involved. The surface of the intestines is found swollen and hyperemic, and there is a thick layer of mucus on the surface. The lymph-follicles, both solitary and agminated, may reveal some hyperplasia and become prominent, and even slight ulceration of the lymph-follicles may be present. Microscopically, the epithelial cells are swollen and granular, many being desquamated from the basement membrane. There may be some leukocytic infiltration into the submucosa, and proliferation of the lymph-nodes. The blood-vessels will be seen to be engorged. In some instances catarrhal inflammation can not be demonstrated at the autopsy.

Symptoms.—Two forms may be recognized clinically, the *acute* and the *chronic* varieties, the most important symptom of either variety being diarrhea, which may indeed be the only manifestation of the disease. The condition may even exist without diarrhea, especially if the jejunum be alone affected. The stools vary greatly in character, the color depending chiefly upon the amount of bile. If bile is present in large amounts, the stools may be dark brown; in less amounts, the stools are light yellow; and if bile be absent, they are of a grayish-white tint. They are thin and watery, and occasionally pultaceous. If portions of undigested food are seen in them, with small amounts of flakish mucus, the condition is known as *lienteric diarrhea.*

Microscopically, the stools contain micro-organisms, crystals of phosphate of lime, cholesterin, Charcot-Leyden crystals, epithelium, and mucus.

Abdominal pain is frequently present in the acute variety. It may be colicky in nature, and if the colon be involved, tormina and tenesmus are present. Some tympanitis occurs. Borborygmus is noted. Often in the acute forms vomiting is a symptom, and a subfebrile temperature is not infrequent. Anorexia, with a dry, coated tongue and much thirst, is present in the majority of cases. The number of stools vary from four to twenty or more in the course of a day. The acute attack may last from two or three days to a week or more.

The *chronic variety* frequently follows the acute form, but as has been pointed out in the etiology, may be an independ-

ent affection. Diarrhea, with or without colic, is a symptom, especially diarrhea of the lienteric character. In the chronic form the general nutrition of the patient suffers, and there may be anemia. If there be marked diarrhea, the blood may show concentration, the erythrocytes being relatively increased.

If the inflammation is confined to the small intestine, diarrhea is a less prominent symptom than if it occur in the colon. Pain is usually of a colicky nature, the stools are yellowish-green in color, and often contain particles of undigested food, and do not contain much mucus, blood rarely being present in the stools. If the inflammation be confined to the large bowel, there may be tenderness over the abdomen along the line of the colon. Diarrhea is more apt to be marked, tenesmus prominent, and the stools contain much mucus. Fever, if present, is subfebrile in range. It is not possible to diagnosticate inflammations limited to certain areas, such as the duodenum, jejunum, or ileum.

Prognosis.—The prognosis in the majority of cases is favorable, the exceptions being where the disease is due to poison. The secondary forms, due to chronic cardiac, renal, or hepatic disease, and the cachexias, are not usually amenable to cure.

Treatment.—The prophylaxis consists in the avoidance of tainted food and impure drinking-water. Attempts should be made to remove the causes in the secondary forms. Rest in bed in the severer forms is important. Very little food should be given, this consisting principally of boiled milk and concentrated meat juices. Vegetables and fruit should be withheld. The salts of bismuth in large doses, from fifteen to thirty grains, four or five times daily, combined with small amounts of opium, are useful. Preceding the administration of the bismuth salts, a brisk purge may be given; this may consist in the administration of calomel or castor oil, followed by salines. In conditions in which much mucus is present, enteroclysis is of value. For the relief of pain, opium suppositories may be given. For the thirst, small pellets of ice may be grateful, large quantities of fluid being avoided. If vomiting be a prominent symptom, food must be entirely withheld. In cachectic individuals stimulation by brandy is of use. A change of climate, especially in summer, as a residence at the seashore or the mountains, is often beneficial in protracted cases.

CROUPOUS ENTERITIS.

Synonym.—Diphtheritic enteritis.

Etiology.—This follows the administration of mercury, lead, or arsenic, or it may be secondary to such diseases as pneumonia, pyemia, septicemia, and enteric fever, or some chronic diseases such as Bright's disease, cirrhosis of the liver, and carcinoma. The pseudomembrane caused by the Klebs-Löffler bacillus sometimes occurs in the intestines, and this should be considered as true diphtheria. Croupous enteritis has been known to follow appendicitis. A fibrinous exudate may be found upon any part of the mucous coat in the small or large intestine. The colon, particularly near the ileocecal juncture, is frequently involved. The membrane is grayish-white in color, varies in thickness, and usually appears in patches.

Symptoms.—The symptoms are not usually distinctive; they are generally pain and diarrhea, the stools frequently containing bloody mucus, and often pieces of pseudomembrane. There may be tenesmus. Fever may or may not appear during the course of this affection, or the disease may run its course without apparent symptoms.

Treatment.—The treatment is symptomatic. It is necessary to relieve pain by means of opium, and to carefully restrict the diet. A laxative may be administered from time to time when tenesmus is marked.

PHLEGMONOUS ENTERITIS.

Synonym.—Suppurative enteritis.

This is a rare affection, and frequently occurs with a corresponding lesion in the stomach. It is also associated with intestinal obstruction, such as intussusception, strangulated hernia, also from infected emboli, etc. It may accompany carcinoma. Abscesses will be found in the submucosa, and when they rupture into the lumen of the bowel a small ulcer is produced.

Microscopically, leukocytes and round cells will be found infiltrated in the coats of the intestines, also necrotic areas and dilated blood-vessels.

ENTERITIS OF CHILDREN.

Synonyms.—Summer diarrhea; infantile diarrhea.

Etiology.—Clinically, three forms have been described—*acute dyspeptic diarrhea, cholera infantum*, and *acute enterocolitis*. The disease occurs more frequently during the hot season, especially in artificially fed children, between the ages of six and eighteen months. The disease appears most often among the poorer classes.

Pathology.—The mucous membrane of the large and the small intestine usually shows catarrhal inflammation. The agminate and solitary lymph-follicles are prominent in many of the cases, and in the more chronic varieties superficial ulceration may even occur. A fibrinous exudate on the mucous coat is rarely found. This when present generally affects the lower part of the ileum and the colon. In many instances the spleen is enlarged. The blood shows anemia, and as a result of the profuse diarrhea the liquid parts of the blood are decreased, the erythrocytes relatively increased (blood concentration). Various forms of micro-organisms are associated with the diarrheas of children. Booker has described a number of varieties of the bacillus coli communis. The bacillus lactis aerogenes is by many considered an important factor in the causation of this affection. The bacillus proteus vulgaris and others are also associated with these diarrheas.

ACUTE DYSPEPTIC DIARRHEA.

The disease usually begins with slight restlessness and an increase in the number of stools, which contain undigested food and curds, and are very often offensive. Occasionally, the disease begins abruptly with abdominal colic, pain, vomiting, and fever, the temperature often reaching 104° F. or 105° F. These symptoms may be preceded by convulsions. Upon palpation the abdomen is painful, and the child's thighs are frequently flexed upon the abdomen. The stools may be grayish or greenish yellow, mixed with undigested food and milk curds. In older children these attacks often follow the ingestion of unripe fruit or tainted milk. This condition may precede the onset of some of the specific fevers, especially those occurring during the hot months. In a weak child even mild attacks may prove fatal. In older and well-nourished children, with proper treatment, the attacks are most often curable.

CHOLERA INFANTUM.

This condition is not nearly so common as was formerly supposed. According to Holt, it occurs in but two or three per cent. of all the summer diarrheas. It arises in the hot months, and most frequently in artificially fed children that have had some previous bowel derangement. The important symptoms are vomiting, profuse diarrhea, and tendency to collapse. The disease often begins with persistent vomiting, aggravated by attempts to take food or drink. At first the stools are very offensive, containing fecal matter, brown or yellowish in color, later becoming thin, watery, and serous, and losing their odor completely. The surface temperature may be low, but the rectal temperature will always show high fever. The child soon manifests the symptoms of collapse, with marked prostration. The eyes are sunken, the features pinched, the fontanels depressed, and the skin pale and ashy. Often there is cyanosis, and delirium and restlessness may be present during the earlier period of the attack; later the child becomes dull and stupid, and passes into coma. Cheyne-Stokes respiration may be present. If the child recovers, fever, vomiting, and diarrhea cease, and the patient is able to take food and retain it. The surface temperature becomes normal and the rectal temperature falls. The pulse, which has been extremely rapid throughout, becomes slower and fuller. Should the attack terminate fatally, the child often dies in coma, with or without convulsions.

ACUTE ENTEROCOLITIS.

The attack may follow acute dyspeptic diarrhea, the symptoms of the latter becoming aggravated, the temperature rising, the pulse becoming more frequent, the stools showing traces of bloody mucus, and the abdomen becoming distended and tender in the line of the colon. There may be vomiting, but it is not so apt to occur as in cholera infantum. If recovery takes place, the diarrhea ceases, and the disease may be over in from two to three weeks. On the other hand, the disease may become subacute, the fever subsiding, but the diarrhea continuing for some time. Another variety occurs, in which there is an intense intestinal inflammation, the symptoms resembling acute dysentery. This form commonly attacks older children. There is tormina and tenesmus, with great prostration, and the disease may terminate fatally in forty-eight hours.

Prognosis in Enteritis of Children.—As has already been mentioned, even simple diarrhea in hot weather, attacking artificially fed children, may be a serious disease, much more so cholera infantum and acute enterocolitis. Recovery often takes place from the two latter conditions under favorable hygienic surroundings, good food, and proper treatment.

Treatment.—Hygienic treatment is of great importance. A change from the hot city to the mountains or seashore is often sufficient to restore the child to health. If this can not be done, open parks or trips upon the water should be suggested. *Fresh air is indicated in all instances.* Cool bathing is of decided benefit, especially if the temperature of the child should rise to 102° F. Iced cloths to the head and abdomen, or injections of ice-water into the bowel, have been followed by good results. In all cases wherever possible, a wet nurse should be procured for a bottle-fed child. If there is much vomiting, it is well to withhold food for some little time. Small amounts of water or small particles of ice should frequently be given. Modified milk is an excellent food for children suffering from summer diarrhea. Egg-albumen is also of use. Where modified milk can not be procured, sterilized milk may be substituted. Mutton, chicken, and beef broths may be given in small quantities from time to time. A laxative should be administered at once. This may be followed by any reliable intestinal antiseptic, such as naphthalin, salol, resorcin, the salicylates, and the salts of bismuth. These must be given in comparatively large doses. Opium should, if possible, be avoided. Alcohol in some form is often necessary for stimulant.

MUCOUS ENTERITIS.

Synonyms.—Mucous colitis; membranous enteritis.

Etiology.—Attention was first called to this disease by Mason Good, in 1825, since which time numerous observations have been recorded. The disease consists in characteristic colic-like abdominal pain, with peculiarly formed stools, consisting largely of mucous masses. The disease is much more common among women, some authorities having put the percentage as high as from 80% to 90%. In the female sex, hysteria and neurasthenia are frequently associated with this affection. When the disease attacks males, they usually belong to the class known as hypochondriacs. In all cases there is a long-standing history of constipation.

Pathology.—The mucous membrane of the colon reveals upon the surface a very tenacious coating of mucus, which sometimes separates as a tubular membrane. Microscopic examination of the stools reveals them to be made up of mucus, many epithelial and pus-cells, and various micro-organisms. In some cases the casts are not present, but instead stringy, ropy mucus is found. The inflammation of the coats of the large bowel is not extensive, and the mucus may separate without leaving a permanent lesion.

Symptoms.—The onset of the disease varies greatly; occasionally it may be insidious. On the other hand, the disease may develop abruptly with the appearance of characteristic symptoms which consist in colic-like abdominal pains and the appearance of mucous masses in the stools. The pains may be exceedingly severe, and are usually felt in the epigastrium and in the left iliac fossa. Occasionally the entire abdomen may be affected, so that the pain may even be noted in the bladder and in the genitalia, or the pain radiates into the left leg, accompanied by considerable tenesmus. The characteristic stools are passed, followed by cessation of the pain, which may be for a longer or shorter interval. The pain may return several times in the day, or may only occur for a week, or for a month or so. Preceding the attack there are anorexia, constipation, and general nervous symptoms, usually with great mental depression. Fever is absent; the pulse is but slightly accelerated.

Prognosis.—As regards life, the prognosis is good. The attack may occasionally cease and not return; in the majority of cases, however, the disease is chronic, and not amenable to cure.

Treatment.—Lately, the condition has been assumed to be a neurosis. Irrigation of the bowel with a normal salt solution has met with success. Occasionally, it is necessary to give castor oil, or a saline or other cathartic during the stage of obstinate constipation. If the pains are severe, sinapisms to the abdomen, hot baths, and opium are necessary. The underlying nervous condition should always be treated, great attention being given to the patient's general hygiene.

INTESTINAL ULCERATION.

Any part of the intestinal tract may be the seat of ulceration, no part of the body revealing ulceration more commonly. Ulceration may result from necrotic changes. Simple duode-

nal ulcers are not infrequent. Duodenal ulcer also arises from extensive superficial burns. Thrombosis and embolism cause intestinal ulceration, and amyloid disease also produces this condition. Ulceration from inflammatory processes may occur, the most common being of the catarrhal variety. They may be follicular, or stercoral which occur in cases of long-standing constipation. Ulceration may occur in acute infective processes, such as enteric fever, dysentery, diphtheria, anthrax, pyemia, erysipelas, and variola. Ulceration due to chronic infectious disease is met with in syphilis and tuberculosis. It takes place in the course of constitutional diseases, such as gout, scurvy, and leukemia. It results from toxic conditions, and from malignant diseases.

Symptoms.—The most constant symptom of intestinal ulceration is diarrhea, but this may be absent when the ulceration is situated high up in the small intestine, or if it be very limited. Constitutional symptoms vary greatly, depending upon the cause of the ulceration. The stools frequently contain pus, epithelial cells, shreds of tissue, bacteria, mucus, and sometimes blood and undigested particles of food. These findings depend upon the seat and character of the ulceration. Pain also varies as to character and distribution: it may be limited, or, if the colon alone is involved, it may correspond to that situation. Perforation and hemorrhage may result from ulceration. This is more common in the acute varieties than in the chronic.

Treatment.—The condition should always be considered as a symptom, the underlying disease producing the ulceration being treated.

CHOLERA MORBUS.

Definition.—A disease characterized by severe abdominal pain, often colicky in nature, with vomiting, purging, and muscular cramp.

Synonym.—Cholera nostras.

Etiology.—The disease occurs most often in the summer months, being favored by bad hygienic surroundings, the eating of unripe fruit and vegetables, and exposure to cold and wet. It may occur at any age and in either sex.

Pathology.—No constant anatomic lesions are present. Most commonly, catarrhal changes of the intestinal tract will be found after death, but in some instances these are absent. Various forms of micro-organisms are associated with this condition, but no specific one has as yet been discovered.

Symptoms.—The onset is abrupt, with severe abdominal pain, nausea, vomiting, and diarrhea. At first the vomited material may contain partly undigested food, later becoming mixed with bile and mucus. The stools at the onset are fecal in character, and many evacuations may take place in the course of a few hours, after which time they lose their fecal character and become serous, not unlike the "rice-water" discharges of true cholera. There is tenderness upon pressure over the abdomen along the region of the colon. Fever is almost invariably present, and it may range from 100° F. to 106° F. The extremities may be cold, although the rectal temperature is raised. The pulse is rapid and feeble, the face becomes pale, pinched, and cyanotic, the urine is scanty and high colored, often containing albumin; and in extreme cases anuria may be present. Cramps in the extremities are common. Thirst is often extreme.

Differential Diagnosis.—The disease may closely resemble Asiatic cholera, and can only be positively differentiated by a bacteriologic examination of the evacuations.

Prognosis.—The disease is rarely fatal. The duration of the attack is from a few hours to several days.

Treatment.—Absolute rest in bed is important. The diet should be restricted, and it is preferable that no food be given during the early course of the attack, especially while vomiting is pronounced. When food is necessary, it should consist of sterilized milk and animal broths, given in small amounts. Local sinapisms or hot turpentine stupes are of great benefit in relieving the pain and in allaying vomiting. It may be good practice at the onset of the disease, especially if there is a suspicion that it is due to improper food such as unripe fruit, and so on, to give a brisk purge. For the thirst, small pellets of ice are of benefit. The remedy for the condition is a hypodermic of morphia. It is rarely necessary to repeat the injection. Later, large doses of the bismuth salts, with opium, are of use. Solid food should only gradually be resorted to.

ENTERORRHAGIA.

Synonym.—Hemorrhage from the bowel.

Etiology.—The condition arises in severe acute catarrh of the bowel, trauma, new growths, cancer, embolism, hyperemia, congestion due to diseases of the heart, lungs, and liver, volvulus, and it results from incarcerated hernia and from obsti-

nate constipation, producing inflammation, from parasites, particularly the anchylostoma duodenale. Local diseases of the bowel, aneurysms, arterial or venous, and foreign bodies, particularly cholelithiasis, will produce the condition. In some infectious diseases, particularly tuberculosis of the intestines; anemic conditions, leukemia, scurvy, purpura, hemophilia, severe jaundice, yellow fever, poisoning from phosphorus, malaria, amyloid diseases of the blood-vessels, erysipelas, enteric fever, dysentery, vicarious menstruation (?), and in melena neonatorum, this condition arises.

Symptoms.—The blood loss may be slight or profuse, *open* or *concealed.* In the open variety, the blood may be either vomited or passed by the rectum. The symptoms depend greatly upon the quantity of blood lost. Small blood loss may give rise to slight or no symptoms. Great blood loss produces collapse. The blood may be passed as pure blood or mixed with fecal material. If the blood be retained in the bowel any length of time, it is apt to be changed by the fluids and passed as a thick, tarry substance.

Prognosis.—The prognosis depends upon the cause and the amount of blood lost.

Treatment.—The treatment consists in absolute rest, and the withholding of food both solid and liquid. Pellets of ice may be given for the thirst. Ice-bags applied to the abdomen, and elevation of the foot of the bed, if the hemorrhage be large, will be found of value. Opium is the most reliable remedy. Small injections of ice-water into the rectum, when the lesion is low down, or iced suppositories may be beneficial.

ENTEROPTOSIS.

Definition.—A condition in which the attachments of the viscera are loosened and there is a dropping of the stomach, intestines, very commonly the transverse colon, the liver, the kidneys, and sometimes the spleen.

Synonyms.—Glènard's disease; visceroptosis.

Etiology.—The condition may be due to a relaxed abdominal wall as a result of repeated pregnancies or ascites, or after the removal of large abdominal tumors.

Symptoms.—It may persist without symptoms, being only detected by physical examination. In other cases symptoms of neurasthenia are present. When occurring in young individuals, neurasthenia is usually pronounced. Physical exam-

ination may reveal displacement of the various organs enumerated. Nephroptosis is commonly met with. Displacements of the liver and spleen are less frequently encountered, and the latter is sometimes quite movable. The colon, especially the transverse, is frequently displaced downward.

Treatment.—A carefully applied abdominal support is often of value. Constipation should be corrected. Tonics may be of value, and the neurasthenia should be treated.

APPENDICITIS.

Definition.—An inflammation of the appendix vermiformis, often giving rise to disease of surrounding structures.

There are no synonyms; the term typhlitis and cecitis mean inflammation of the cecum, perityphlitis meaning inflammation of the serous coat of the cecum. These forms are distinguished with difficulty from appendicitis, but are rarely primary affections, usually resulting from inflammation of the appendix. In enteric fever the cecum frequently reveals the seat of the greatest ulceration, and tenderness in the right iliac fossa is present in a large proportion of cases.

Etiology.—HISTORIC NOTE.—The appendix is a rudimentary, functionless organ, being a relic of the large ancestral cecum. In many of the lower animals the cecum is very large and possesses distinctive functions. Darwin clearly demonstrates the functionless character of the appendix vermiformis in his work, the "Descent of Man," chapter I, page 20, in which he says: "With respect to the alimentary canal, I have met with an account of only a single rudiment, namely, the vermiform appendage of the cæcum. The cæcum is a branch or diverticulum of the intestine, ending in a culdesac, and is extremely long in many of the lower vegetable-feeding mammals. In the marsupial koala it is actually more than thrice as long as the whole body. It is sometimes produced into a long, gradually-tapering point, and is sometimes constricted in parts. It appears as if, in consequence of changed diet or habits, the cæcum had become much shortened in various animals, the vermiform appendage being left as a rudiment of the shortened part. That this appendage is a rudiment, we may infer from its small size, and from the evidence which Prof. Canestrini has collected of its variability in man. It is occasionally quite absent, or again is largely developed. The passage is sometimes completely closed for half or two-thirds

of its length, with the terminal part consisting of a flattened solid expansion. In the orang this appendage is long and convoluted; in man it arises from the end of the short cæcum, and is commonly from four to five inches in length, being only about the third of an inch in diameter. Not only is it useless, but it is sometimes the cause of death, of which fact I have lately heard two instances: this is due to small hard bodies, such as seeds, entering the passage, and causing inflammation."

Also Haeckel demonstrates this in volume II, page 344, of his work, "The Evolution of Man," as follows: "In man, as in most apes, the beginning of the blind intestine alone becomes wide; its blind end remains very narrow, and afterward appears only as a useless appendage of the former. This 'vermal appendage' is interesting in dysteleology as a rudimentary organ. Its only importance in Man consists in the fact that now and then a raisin-stone, or some other hard, indigestible particle of food becomes lodged in its narrow cavity, causing inflammation and suppuration, and, consequently, killing individuals otherwise perfectly healthy. In our plant-eating ancestors this rudimentary organ was larger, and was of physiological value."

Following this line of argument, it is conclusive that the most marked predisposing factor is the *evolutionary tendency*, to be distinguished from the *hereditary tendency* which shows itself from generation to generation. The evolutionary tendency makes its appearance only after changes of environment.

Predisposing Causes.—(1) The most important is the evolutionary tendency, and under this heading various abnormalities must be considered. *Strictures.*—Narrowing of the lumen of the appendix prevents the normal drainage of the tube, establishing favorable conditions for the growth of micro-organisms. This is especially true when the stricture is situated near the proximal end, or at the valve which guards the opening of the appendix into the cecum, called "Gerlach's valve." The strictures may be multiple. In some instances the canal is obliterated. (2) *Blood Supply.*—The blood supply of the organ may be defective, causing anemia, and in this way predisposing the organ to infection. This may also be brought about by the free mobility of the appendix, thereby twisting the blood-vessels in a long meso-appendix, and thrombosis may occur in the artery. Atrophy of the appendicial walls may be present from the diminished blood supply and from the pressure of fecal concentration occupying the lumen. The

latter, however, frequently produces atrophy of the walls. (3) *Position and Size of the Appendix.*—The most common position of the organ is pointing in the direction of the spleen, and next in frequency is its position behind the cecum. It may extend over the brim of the pelvis, or toward the umbilicus. It has been found in the inguinal canal, also in the intussusceptional portion of the colon in cases of intussusception. It fact, it may be stated that the appendix may occupy various positions. The organ varies greatly as to length and diameter; however, it usually measures about seven or eight centimeters.

The histology of the appendix closely resembles that of the cecum. The submucosa is freely supplied with lymph-follicles; the muscular coats are usually not well developed. Occasionally the appendix is absent altogether.

The majority of cases of appendicitis develop in young persons; however, it has been met with in the extremes of age. It is more prevalent in the male than in the female sex. In occupations requiring exertions that produce marked contraction of the abdominal muscles, and hence compression of the abdominal contents, fecal or gaseous matter may be forced into the lumen of the appendix, which may predispose the organ to infection. Diarrhea is also a predisposing cause, especially when there is inflammation of the cecum. Constipation is a predisposing cause.

The appendix may be involved from extension of inflammation. Indiscretion of diet, such as eating highly seasoned foods, overeating, or excessive drinking may produce inflammation. Many of the infectious diseases predispose, especially influenza, diphtheria, enteric fever, tuberculosis, the rheumatic diathesis, and the eruptive fevers. One attack, far from conferring immunity, predisposes to other attacks, and in this disease *relapses are common.*

Exciting Causes.—The most important of the exciting causes are micro-organisms; the most common of these, which produces inflammation, is the bacillus coli communis. Many other micro-organisms, such as the bacillus typhosus, the micrococcus lanceolatus, the staphylococci, the tubercle bacillus, the streptococcus, the bacillus pyocyaneus, and others have been known to be associated with the affection.

Bodies in the lumen of the appendix often act as exciting agents, the most common being the fecal concretions, these causing an erosion of the walls, sometimes with perforation, or allowing micro-organisms to set up inflammation. Foreign

bodies, such as grape seeds, date seeds, apple pits, stones of various fruits, hair, and small pieces of wood, and, in a case described by Deaver, a pin perforated the wall of the appendix have given rise to the affection. Foreign bodies, however, form a very small percentage as direct causative factors of the affection, but are predisposing causes.

Traumatism may be an exciting cause. The appendix may be diseased as a result of extension of inflammation, especially from the cecum.

Pathology.—The pathology of appendicitis may be said to present various phases met with in inflammation. In many cases a *simple acute catarrhal inflammation* is encountered; again, this may be *chronic catarrhal inflammation.* Interstitial inflammation, frequently involving all the coats, is found, or in this form *ulcerative appendicitis* is common, the ulcer being either deep or superficial. In some instances it may perforate. The interstitial inflammation may give rise to *phlegmonous and gangrenous appendicitis*, this usually being preceded by interference with the circulation of the organ. *Abscess formation* may follow. There may be chronic interstitial inflammation known as "*fibroid appendicitis*," or "*appendicitis obliterans*," resulting either from repeated attacks of acute catarrhal or acute interstitial inflammation. This frequently gives rise to the development of stricture, which in turn predisposes to acute attacks. Extensive *retroperitoneal abscesses* often result from appendicitis. These are frequently in the flank, and may form large perinephritic abscesses. The psoas muscle may also be invaded by pus. As a result of necrosis and sloughing of the appendix, this organ may be completely destroyed, and it is not uncommon to find at the operation that a single and small slough represents the remains of the appendix. A fibroid change may begin at the distal end of the appendix and extend toward the proximal extremity, obliterating the lumen known as *appendicitis obliterans* (the term first used by Senn).

The formation of new connective tissue in the walls of the appendix may be so pronounced as to cause atrophy of the muscular, submucous, and mucous coats, and if the internal pressure be marked, dilatation of the lumen results, with thinning of the walls.

As a result of acute inflammation, the peritoneum may be involved by extension, or the serous membrane may become inflamed as a consequence of perforation. The peritonitis which follows appendicitis may be *local* or *general.* As a rule,

the inflammation is confined to the right iliac fossa, and abscess formation not infrequently results when the inflammation is confined to this area; however, if the appendicitis be situated so that the greatest point of inflammation be away from the right iliac fossa, abscess may arise in this situation, and such abscesses are sometimes met with in the pelvis or in the flank. Again, the appendix has been known to perforate into the bladder. The cecum often is secondarily involved in acute infective appendicitis, particularly if the organ be situated in close relation.

Hemorrhage sometimes results, either from necrosis of the appendicial arteries, or from ulceration of the internal iliac or the deep circumflex artery; superficial pylephlebitis may arise from inflammation of the appendix, this inflammation not infrequently extending into the liver, or infected emboli may be carried into the liver and produce abscesses in this organ.

Symptoms of Acute Appendicitis.—The onset is sudden. There is pain in the abdomen, which at first may be general or centered around the umbilicus, and occasionally in the epigastrium and in the left or right iliac fossa. In many of the cases the pain is confined to the right iliac fossa and directly in the region of the appendix. If the pain is general or confined to various parts of the abdomen, it soon localizes itself to the right iliac fossa, usually within the course of twenty-four hours. It is paroxysmal, or intermittent, and colic-like. In some instances it is sharp and intense, particularly if it involves the peritoneal coat; this has been called *serous membrane pain.* In other cases, especially when the serous coat has not been involved, the pain is dull, and called "*connective-tissue pain.*" Pain is the most constant of all the symptoms. It may be preceded by chilliness; frequently nausea and vomiting mark the beginning of the attack. Fever rapidly follows the onset of the disease, which is usually moderate, varying from 100° F. to 103° F.; however, it may be absent in some cases. When local suppuration results, the fever may be marked and of a septic type. When general peritonitis sets in, the temperature may be of the septic type, or in some instances remain normal. The frequency of the pulse generally corresponds to the degree of the fever. The surface temperature is sometimes higher over the right iliac fossa than the left. Constipation is commonly present in this disease, but in some instances there is diarrhea. The tongue is coated posteriorly with a moist fur. The urine gives the characteristics of febrile conditions. It

may be albuminous; frequent micturition is a symptom in some cases.

Physical Examination.—*Inspection.*—The facies shows suffering. The expression is anxious, the patient carefully noting and observing his condition. The position of the patient is often characteristic—the right thigh is most frequently partially flexed upon the abdomen, while the left leg is extended. On examination of the abdomen, slight distention is noted. The breathing is shallow and thoracic.

Palpitation.—On palpating the abdomen the right rectus muscle will be found rigid; sometimes this rigidity also exists in other abdominal muscles. It is present if the patient comes under observation early in the course of the attack; later, it commonly disappears. Tenderness is as constant as pain. It is usually localized to "*McBurney's point*" (a point at the insertion of the line drawn from the anterior superior spinus process of the ilium to the umbilicus, and another along the right edge of the rectus muscle). This tenderness is noted either upon superficial or deep palpation; however, the point of greatest tenderness is not always in the region described by McBurney, but in other situations, and may be said to correspond to the point of the maximum intensity of the inflammation. Pressure upon the opposite side frequently produces pain in the region of the appendix. If the primary rigidity has subsided, a tumor is often palpable in the right iliac fossa, which may vary in size. In some instances the appendix may be clearly made out. Examination per rectum and per vaginam frequently reveals tenderness in the inflamed area when pressure is made in that direction.

Percussion.—If a tumor exists there is dullness upon percussion.

Local Abscess Formation.—The symptoms just enumerated may subside, or in some instances they become aggravated. The fever becomes higher and often of a septic type; the pulse rapid and feeble; and chills and sweating may occur. The pain becomes dull, less severe, and often disappears. Upon *inspection*, in some cases local redness and edema are noticed. Upon *palpation* the rigidity of the abdominal muscles is not present, and a large mass may be clearly demonstrated, the most frequent situation being in the right iliac fossa lying upon the psoas muscle; however, the abscess may be present in the flank or just above Poupart's ligament, and sometimes in other situations.

Complications and Sequels.—Gangrene and perforation of the blood-vessels and neighboring tissues may occur. Perforation is a frequent complication. It is recognized by signs of collapse, and fall in the temperature, rapid running pulse, great tympany, and obliteration of the lower border of liver dullness. Peritonitis is a serious complication. It is most often local, and limited to the region of the appendix; it may, however, be general. General peritonitis is the most frequent cause of death. Persistent vomiting is an exceedingly serious complication of appendicitis. (For symptoms see Peritonitis, p. 506.) The gravity of this disease lies in the fact that the peritoneum may be involved, and the early symptoms may indicate a widespread infection of this serous membrane. Hiccup is a distressing symptom, and is often due to general peritonitis, and frequently precedes the fatal issue. Pylephlebitis and hepatic abscess are grave complications. The lungs, pleura, and heart are rarely involved. Hemorrhage from involvement of the iliac arteries or veins, or from the very close position of the mesenteric or ovarian veins, may produce a disastrous result from an extension of the gangrenous process from the appendix. Intestinal obstruction is an important complication.

CHRONIC APPENDICITIS.

There may be constant pain of a dull character in the right iliac fossa, persisting for long periods of time without fever or other general manifestations. Tenderness is present in the right iliac fossa. The appendix may or may not be palpable. The pain is sometimes aggravated by taking food.

RELAPSING APPENDICITIS.

After an acute attack the symptoms may subside, and the patient regain complete health. Sooner or later, however, another attack follows, the period of recovery varying greatly, sometimes extending over a period of months or even years. The second attack is generally more severe, but in some instances is of a milder character. Again, recovery may follow, or the attack may prove fatal. A number of these attacks are not infrequent in some cases. *It may be said that after one attack of appendicitis recurrence is the rule.*

Diagnosis.—The direct diagnosis of appendicitis depends upon the sudden onset of the disease, of pain in the right iliac fossa, tenderness in this region, rigidity of the right rectus

muscle, vomiting, fever, and in many instances a palpable tumor.

Differential Diagnosis.[1]—

	Appendicitis.	*Biliary Colic.*	*Renal Colic.*
Pain.	Colicky, radiating over the lower part of the abdomen toward the umbilicus; tenderness over "Mc Burney's point."	Radiating over the upper half of the abdomen and toward the right shoulder; tenderness over the gall-bladder.	Radiating less over the abdomen, but down the ureter to the testicles, head of the penis, and often irritating the rectum; tenderness over the kidney and lumbar region.
Vomiting.	Usual, but not extreme.	Pronounced and persistent.	Present, but not persistent.
Symptoms Relating to the Bladder and Testicle.	Absent.	Absent.	Marked.
Urine.	Normal.	May contain bile.	May contain blood and mucus.
Jaundice.	Absent.	Present.	Absent.

Typhoid fever should not be confounded with appendicitis, for its onset is insidious, marked by pronounced tenderness in the right iliac fossa, a typical temperature curve, and the characteristic eruption, with enlargement of the spleen; however, when perforation in the course of enteric fever is the first symptom brought to the notice of the physician, the diagnosis may be difficult. It must also be remembered that appendicitis may be a complication of enteric fever. Inflammation of the tubes, ovaries, and pelvic peritonitis may closely simulate appendicitis, but the history of the patient and local examination in most cases reveal the true nature of the affection. Hysteria may resemble acute appendicitis, and is sometimes with difficulty diagnosticated from this condition.

Prognosis.—There is much uncertainty as to the prognosis of appendicitis. Much depends upon the involvement of the peritoneum, the prognosis being more unfavorable in this condition. Recurrence of attacks is common. Spontaneous recovery from the obliteration of the lumen of the appendix may occur, but this is infrequent.

Treatment.—This had better be considered a surgical affection. The surgeon should be promptly called. Osler says he is often called too late, but never too early. Rest in bed is important. The diet should be restricted, and

[1] Modified from Loomis-Thompson's System, by McNutt.

nutriment given in small amounts; in many instances the food had better be withdrawn until vomiting subsides. Small pellets of ice are given to allay thirst. Opium may be administered to relieve pain. Ice applied locally is of great value in relieving the pain, and this method had better be practised before the diagnosis is made certain. Purging by calomel and salines should be resorted to very early.

INTESTINAL OBSTRUCTION.

Intestinal Obstruction Due to Strangulation.—This is a frequent form of intestinal obstruction. It may result from adhesions of the peritoneum. Appendicitis causes adhesions. Mesenteric and omental hernia, adhesive tubes, peritoneal pouches (Meckel's diverticulum), and pedicular tumors may cause the condition. It often follows operation upon the abdomen, being due to bands of adhesion. The condition is most frequently met with in males, the greater number of cases occurring in early adult life. The small intestine is more commonly involved than the large.

Intestinal Obstruction Due to Twists and Knots, Called Volvulus.—This is frequently associated with an unusually long mesentery, and a very common site is in the sigmoid flexure, the next in frequency being the cecum. The intestine is usually twisted upon its long axis, thereby causing strangulation. It is more prevalent in males, and between the ages of thirty and forty.

Intestinal Obstruction Due to Strictures and Tumors.—These forms of obstruction usually give rise to symptoms of a chronic nature. The obstruction may be due to congenital narrowing or complete obliteration of the lumen, such as an imperforate anus. It may be due to a cicatricial contraction as the result of ulceration, particularly from syphilis, tuberculosis, and dysentery. Tumors of the intestine not infrequently cause obstruction, particularly carcinoma, papilloma, adenoma, lipoma, and fibroma. Tumors pressing upon the wall of the intestine may give rise to the condition, or in rare instances pressure from the distended coil of intestine pressing upon a neighboring coil may cause the obstruction.

Abnormal contents of the intestine may produce obstruction, such as foreign bodies, particularly coins, pins, needles, false teeth, fruit-stones, and intestinal worms. Some drugs, such as magnesia and bismuth, when taken in large amounts

accumulate in the bowel and cause obstruction. Fecal impaction, gall-stones, and enteroliths may occlude the intestinal lumen.

The symptoms of intestinal obstruction may be divided into acute and chronic.

Symptoms of Acute Intestinal Obstruction.—The onset of the attack is sudden in the majority of instances, no exciting cause being apparent. The first symptom is severe abdominal pain, localized around the umbilicus; however, it may occur in any part of the abdomen. It is constant, and liable to exacerbations. Sooner or later, tenderness of the abdomen develops, with symptoms of collapse, great depression, pallor, feeble pulse, cold sweat over the face, body, and extremities, nausea, and vomiting. Vomiting occurs early; it often consists of the contents of the stomach, then becomes bilious, and finally brownish and offensive, being composed of fecal material (*stercoraceous vomiting*). Constipation is the rule from the beginning; however, there may be an evacuation of the intestinal contents below the seat of the stricture. The belly is distended and tympanitic. The tongue is coated, and thirst is intense. The amount of urine is diminished. There may be some rise in temperature in the beginning, but the temperature soon becomes subnormal as collapse occurs. If the condition remains unrelieved, the symptoms persist, and signs of septic poisoning, which are so common as a terminal event of acute peritonitis, appear. Delirium may be present, but as a rule consciousness is retained to the last. Vomiting persists to the end. The majority of cases, if not relieved, terminate fatally in from six to seven days.

Symptoms of Chronic Intestinal Obstruction.—The onset is gradual and the progression of symptoms irregular. Abdominal pain occurs paroxysmally, often provoked by food, and frequently ascribed to indigestion. The pain is not so severe as in the acute forms. The attacks finally become more frequent, of longer duration, and of increased severity. Some vomiting, constipation, less commonly diarrhea, with more or less uneasiness within the abdomen are present. The vomiting is slight at first, and not persistent, but nausea and disinclination for food may exist throughout. At first the constipation is not absolute, the patient being relieved by laxatives; drugs, however, act with less and less effect, and occasion more pain and vomiting. Occasionally there may be spurious diarrhea, due to catarrh in the bowel excited by retained fecal matter

below the obstruction. It is especially apt to occur when the stenosis is low down in the colon. The tongue is coated with a white fur and the breath is particularly offensive. The amount of urine is normal, and the temperature is most often undisturbed. The belly becomes more and more distended as the disease advances, a tumor often being discernible. This may be due to the accumulation of feces above the obstruction. Visible coils of the intestine may be seen in active movement through the apparently thin abdominal walls. Upon movement of these coils of intestine, pain is aggravated. Borborygmi are common. Death in these cases may occur in six months after the onset of the symptoms.

Diagnosis.—The diagnosis of intestinal obstruction must be made from obstruction resulting from hernia, the latter in some instances being concealed. Examination of the patient is necessary to reveal the condition. Intestinal obstruction may be confused with appendicitis. In the latter, fever is a pronounced symptom, pain is localized to the right iliac fossa, constipation is not complete, and vomiting occurs early and is not likely to become stercoraceous. There is also less distention of the abdomen.

Differential Diagnosis.—

Obstruction in the Small Intestine.	*Obstruction in the Large Intestine.*
The symptoms at the onset are most often acute.	Most often chronic; volvulus of the sigmoid flexure very acute.
Pain appears earlier, is more pronounced, and more severe.	Pain is less marked.
Vomiting appears earlier; is more distressing, and more persistent; vomited matter is more copious, and is influenced by taking food.	Vomiting is more irregular; may be slight; rarely stercoraceous.
Constitutional disturbances most marked; shock frequent.	Constitutional disturbances slight; shock infrequent.
Meteorism occurs late, and is seldom of high grade.	Meteorism occurs early, and is well marked.

Prognosis.—In acute intestinal obstruction the prospect for relief is very slight, except in the rarest instances. All cases, if not treated, terminate in death. Death may occur in from two to seven days, and in the subacute form in from seven to thirty days. Cases of chronic obstruction may extend over a period of many months. Ulceration, perforation, and general peritonitis may be the cause of death, or a coil of the gut may become gangrenous. In a small proportion of cases death is due to septic pneumonia.

Treatment.—The treatment is surgical. Opium should be

given to relieve the pain. If vomiting be persistent, the stomach-tube may be used, the stomach-contents siphoned, and the organ washed. Purgatives should be avoided. Hot turpentine stupes may be applied to the abdomen, usually giving relief. Ice-bags may allay the pain, but when collapse occurs, ice should not be used. In chronic obstruction opium is necessary for the pain. Rectal feeding may be resorted to. *Surgical interference sooner or later becomes imperative.*

INTUSSUSCEPTION OF THE BOWELS.

Definition.—"Intussusception consists in the entrance of one portion of the intestine within another by an infolding of the bowel so that the external fold insheathes the inner." (Lyman in Loomis-Thompson's System.)

The upper portion of the bowel is almost always invaginated into the lower, so that the lowermost portion insheathes the upper portion. Upon cross-section of the telescoped bowel, three layers of the intestine are found—the outermost, or receiving layer, called the *intussuscipiens;* the middle layer, or returning portion, and the innermost portion, called the entering layer. The middle and internal layers together are called the *intussusceptum.*

Etiology.—Increased peristalsis. Sometimes it results from normal peristaltic movements. It may arise from spasm or paralysis of the intestines, and it is sometimes associated with carcinomatous growths, or inflammatory conditions of the intestine. Obstinate constipation and chronic diarrhea seem to predispose. It has been known to follow trauma. It is most frequently met with in children, and is more prevalent in the male than the female sex.

According to Leichtenstern, who recorded 593 cases of intussusception, 131 cases occurred in the first year of life; among these, 80 took place from the fourth to the sixth month; 49 cases occurred between the second and the fifth years of life. Pilz recorded 162 cases in children, in which 91 took place in the first year of life, 71 occurring between the second and the fourteenth years of life.

Pathology.—Intussusception may occur at any part of the intestine, but is most common in the ileocecal or ileocolonic region, involving in this position the ileum and the cecum. It may involve the ileum alone, when it is called ileac invagination, or the jejunum, when it is called jejunum invagination, or

the colon alone, when it is called colonic invagination. Upon postmortem examination the condition of the intussuscepted bowel is quite characteristic in its appearance. If death result early from shock, the involved area simply reveals the telescoped bowel; most frequently, the affected portion is swollen and reddened, the peritoneum revealing inflammatory changes. The bowel may be gangrenous, and hemorrhage into the lumen of the intestine is frequently found. The intussusceptum usually shows more marked inflammation than the intussuscipiens, for the reason that the blood supply is more or less cut off. The innermost portion of the bowel may reveal marked sloughing and complete separation in some instances. Recovery has resulted from the occurrence of separation of the telescoped bowel, healing of the bowel taking place at the point of separation with the lower end of the intestine. Perforation and general peritonitis may occur.

Symptoms.—The condition begins suddenly in the midst of health, with pain, usually of a colicky nature, localized to the abdominal region, and the abdomen becomes distended. This may occur during sleep. In rare instances the onset may be more insidious. In children it may be ushered in by a loud cry or by convulsions. The pain may be localized at a particular point and radiate from there. Vomiting is a frequent, although not an invariable, symptom. The vomited material may consist of fecal matter. Constipation is the rule, accompanied by considerable tenesmus. Frequently in the stool, blood and mucus are present. Soon symptoms of collapse develop; the pulse becomes small, rapid, and feeble. The vomiting recurs, bloody mucoid discharges from the bowel may continue, and the abdomen becomes tympanitic. These symptoms may soon be followed by death. In some cases remissions may occur and the condition become more or less chronic. In these cases the pain is usually milder, and vomiting and bloody diarrhea may be absent altogether. Complete anorexia is the rule. On examination, the abdomen is often found somewhat distended, tense, and painful, and a sausage-like tumor can be made out; and sometimes upon rectal examination the lower border of the intussusception can be felt.

Diagnosis.—The diagnosis depends upon the finding of the sausage-like tumor upon palpation, accompanied by tenesmus and bloody stools.

Prognosis.—The prognosis is unfavorable, about 70% of

the cases proving fatal. Spontaneous recovery sometimes results from the separation of the invaginated portion of the bowel, but in some cases this part may be discharged in small fragments.

Treatment.—The results of surgical treatment are more favorable than those of medicinal measures. Opium is necessary to relieve the pain and to check peristaltic action of the intestine, thereby preventing further invagination. Distention of the colon by means of warm water slowly introduced may prove of value in relieving the invagination. Inflation of the intestine by means of air is sometimes practised. If the invagination be low down in the colon, a rectal bougie carefully introduced may relieve the condition. Ice-bags may be applied locally to allay pain. Food should be withheld, and ice given to relieve thirst.

HEMORRHOIDS.

Synonym.—Piles.

Etiology.—Varicose enlargements of the hemorrhoidal plexus of veins are known as hemorrhoids. This condition may result from local pressure, frequently from constipation, tumors of the rectum, growths from without, enlargements of the uterus and ovaries, sometimes from an enlarged prostate, and from stricture of the rectum, particularly in syphilitic disease. Interference with the portal circulation, especially in atrophic cirrhosis, almost constantly causes hemorroids. Diseases of the heart and lungs may prevent the return of the venous blood and cause stagnation in the hemorrhoidal plexus. Hemorrhoids are commonly met with in middle life. Children rarely suffer from piles. Free indulgence in food and lack of exercise predispose to hemorrhoids. The affection is also met with in gout and obesity, and is more frequently found in men than in women.

Pathology.—Hemorrhoids may be situated above the sphincter ani muscle, when they are called *internal* hemorrhoids. When they are situated below, they are called *external* hemorrhoids. They may be located in either position, and frequently in both. When existing externally, they appear as irregular links or discs surrounding the anus, are wart-like, and of a bluish color. They vary as to size. Internal hemorrhoids are usually broad and flat, and sometimes are pedunculated, and may protrude through the anus. Inflammation of the mucous membranes surrounding the hemorrhoids is almost

always present. Hemorrhoids may undergo ulceration and suppuration, and frequently hemorrhage results from them. Fistulæ may be formed. Thrombosis may occur in these veins.

Symptoms.—The symptoms vary greatly, depending upon the extent. Often they cause great annoyance, accompanied by pain of a burning or smarting character, with a sensation as if the rectum were occluded by a foreign body. The pain is increased during defecation, by horseback-riding, by excessive exercise, or by a long-continued sitting posture. Sometimes nausea, vomiting, and palpitation of the heart accompany these symptoms. A mucopurulent bloody discharge is frequently met with as a result of internal hemorrhoids. Sometimes hemorrhage may be quite profuse. When hemorrhoids prolapse and strangulation results, pain is intense.

Diagnosis.—The diagnosis of hemorrhoids is made by inspection and palpation, the rectal speculum often being of value in the diagnosis of internal hemorrhoids.

Prognosis.—The condition does not prove fatal unless accompanied by complications such as septic infection. The symptoms usually subside after the removal of the cause.

Treatment.—Constipation should be relieved and the diet carefully regulated. The cause, if possible, should be removed. It must be remembered that atrophic cirrhosis of the liver is usually accompanied by hemorrhoids. Surgical interference may prove of value if the condition is purely local, but not if arising from obstruction of the portal circulation. The affected area should be carefully cleansed, especially after defecation. Local application of ice is of benefit in relieving pain, inflammation, and hemorrhage. Astringents, such as tannic acid and nitrate of silver, may be of value in allaying pain. An ointment containing carbolic acid, cocain, and belladonna, or one of tannic acid or gallic acid and opium, is of great value. Hot applications are sometimes most soothing to the patient.

TUMORS OF THE INTESTINES.

BENIGN TUMORS.

Of the benign epithelial tumors, *adenomata* are by far the most frequent. They vary greatly in size, and the most common situation is in the rectum immediately above the anus; they are found in other parts of the intestines, but are rare. They most often appear in children between the ages of four and

seven years (Nothnagel). The surface frequently indicates ulceration, and hemorrhage may take place. These tumors not infrequently terminate in true carcinoma; that is, when the epithelial cells rupture through the basement membrane.

Papillomata occur near the end of the rectum and the lower part of the ileum. Of the connective-tissue tumors, *fibromata* and *lipomata* are most frequently found. They may appear singly or may be multiple, and vary in size from a pea to a large apple. As in papilloma, the most frequent seat is in the rectum, and next in frequency in the ileum, and they may also appear in other parts of the intestinal tract.

Myomata and *fibromyomata* are less common, but have been observed in the intestinal tract; *angiomata* are exceedingly rare.

Symptoms.—Symptoms are not characteristic, and unless the tumors reach a large size, symptoms may be entirely absent. Enterorrhagia occurs, especially with angioma, but may also take place with other benign tumors. If enterorrhagia take place in the earlier periods of life, tumors of the bowel should be suspected. Symptoms of obstruction may also appear. Benign tumors can not, as a rule, be palpated through the abdominal wall. The tumor may cause death from hemorrhage, obstruction, or invagination. It must always be remembered that carcinomatous change may occur in the epithelial type.

Treatment.—The treatment consists in relieving the obstruction, or, if hemorrhage take place, in treating that condition.

MALIGNANT TUMORS.

Carcinoma.—This is the most common tumor of the intestines. It is more often found in the male sex, and after the fortieth year of life. As to the locality of carcinoma, it is most frequently met with in the rectum, next in frequency in the colon. It also occurs in the duodenum, particularly at the papilla of the common bile-duct; it also occurs in the jejunum, ileum, and cecum.

The statistics covering twenty-four years, from 1870 to 1893 inclusive, from the Pathological Institute of the Allgemeines-Krankenhaus in Vienna, collected by Nothnagel, are as follows: Out of 41,838 autopsies, 343 carcinomata of the intestines were found; affecting the duodenum, 7 cases; the ileum, 10; the colon, 164; the rectum, 162. The small intestine is much less frequently affected than the large. They are nearly always

primary. The most common variety is the adenocarcinoma. Its most common seat is in the lower part of the rectum. The scirrhous carcinoma is very rarely found in the intestines. Encephaloid carcinoma is also found in the intestines. Squamous cell epithelioma may be met with at the lower end of the rectum.

The cancers of the intestine vary greatly as to size. They are commonly found as irregular masses projecting into the lumen. Sometimes the cancer encircles the entire lumen. They are prone to ulceration, and when this is extensive, perforation and fistulous communications may occur. As a result of the ulceration, hemorrhage is not infrequently met with. Colloid degeneration may be encountered. These tumors are rarely of large size. All the coats of the intestine are usually involved. Fistulous communication may be established with other parts of the bowel, the stomach, bladder, vagina, and even in some instances through the abdominal wall. The most frequent seat of metastasis is in the lymphatic glands and the liver.

Symptoms.—The disease may run its course without characteristic symptoms, excepting the cachexia and anemia so common in malignant diseases. In rapidly growing cancer fever commonly occurs; otherwise the temperature may be normal or subnormal. The condition sometimes appears with symptoms of occlusion of the bowel, preceded by obstinate constipation in the majority of cases. Hemorrhages from the bowel are likely to take place. The character of the stools varies; they may be soft, putty-like, scybalous, or ribbon-shaped. Pain is a common symptom. It may be local or it may radiate. It becomes general when peritoneal involvement occurs. It varies in intensity from a dull, unpleasant sensation to severe, sharp, colicky pain. Bodily wasting becomes marked as the disease progresses. If the situation of the cancer be near the opening of the common bile-duct, symptoms of biliary obstruction are met with; jaundice then becomes a prominent symptom. The blood reveals a marked decrease in the hemoglobin, and a decrease in the erythrocytes, accompanied by malignant leukocytosis.

Physical Examination.—The abdomen is usually scaphoid, but when symptoms of obstruction are present, it may be distended. In rare instances the tumor may be visible as a protruding mass, most frequently in the left iliac fossa. Upon palpation, a relaxed abdominal wall is met with, and an irreg-

ularly oval or rounded mass, painful upon pressure, is felt. If adhesions are not present, the mass is somewhat movable, and when metastasis into the lymphatic glands has occurred, small nodular masses are sometimes palpable. Sometimes the mass can be palpated per rectum and per vaginam. Percussion gives dullness over the tumor.

Diagnosis.—The diagnosis of cancer of the intestine depends upon the age of the patient, and in many instances upon a hereditary tendency, the cancerous cachexia, loss of weight, pain, and the detection of the tumor upon palpation.

Prognosis.—Death from carcinoma of the intestines may take place during the course of a few weeks or months or not for several years.

Treatment.—When the diagnosis is made early, surgical interference may be of some avail, but usually the condition must be treated palliatively. A light, easily assimilated, nutritious diet should be given. For the pain, opium is indicated. Careful attention should be paid to the condition of the bowels; light purgation and enemata are frequently useful. Cannabis indica is sometimes of value. When symptoms of obstruction are present, lavage and rectal feeding are necessary.

Sarcoma of the Intestines.—Primary sarcoma of the intestines is rarely encountered. The disease is usually met with in early life, but may occur in middle or advanced periods. These tumors spring either from the submucosa or from the deeper layer of the intestine. They may give rise to symptoms of obstruction. Secondary sarcomata are also found in the intestines.

DISEASES OF THE PERITONEUM.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneum. It may be local or general.

ACUTE GENERAL PERITONITIS.

Etiology.—The most frequent mode of infection is through the intestines and from the female sexual organs. Various forms of ulcer may produce peritonitis, such as typhoid, tubercular, diphtheric, decubital, peptic, syphilitic, and appendicial

inflammation. Malignant diseases may produce acute peritonitis. It also arises from forms of intestinal obstruction, such as volvulus and stricture, occasionally from traumatic rupture of the intestine, and from impacted feces. Less commonly, the inflammation may have its origin in disease of the stomach, particularly from gastric ulcer. The gall-bladder and liver may be the source of the infection, and particularly abscesses; and syphilitic disease may give rise to it. It may also be of carcinomatous or echinococcous origin, or may result from the thickening of the interstitial layer from hepatitis. *Peritonitis rarely occurs from inflammation of the liver.* Obstruction of the biliary passages by gall-stones may produce peritonitis. The affection may arise from the spleen, through an infected embolus, or from perisplenitis. Infection from the pancreas is rare. It may result from infective diseases of the kidney. The affection may originate from diseases of the bladder-wall, from the prostate, and the urethra. During the course of erysipelas, especially when it involves the skin of the abdomen, peritonitis may occur. Peritonitis has been noted during the course of acute rheumatic fever and septicemia, rarely during the course of scurvy. The disease may be primary or secondary, most frequently the latter. There are cases due to bacteria, chemic causes, and mechanical causes.

Peritonitis Due to Bacteria.—Various *bacteria* may cause peritonitis, such as the streptococcus pyogenes, the bacillus coli communis, the pneumococcus (diplococcus pneumoniæ), the staphylococcus pyogenes aureus, bacterium lactis, and, according to Nothnagel, the diplococcus intestinalis major and minor, streptococcus conglomeratus, the ray fungus, the bacillus of tetanus, glanders, and diphtheria, the bacillus pyocyaneus and streptococcus pyogenes, the proteus vulgaris, the bacillus of tuberculosis, the gonococcus, Eberth's bacillus (bacillus typhosus), and the bacillus of anthrax. Some of these forms of micro-organisms may cause primary peritonitis without causing inflammation of other structures; most commonly, however, the inflammation of the serous coat is secondary to inflammation of some surrounding tissue, such as the intestine from ulceration, also from inflammation of the pelvic organs and stomach. The three micro-organisms most commonly associated with this inflammation are the streptococcus pyogenes, the bacillus coli communis, and the bacillus tuberculosis. Infection may be single, when due to one group of micro-organisms; or mixed, when resulting from two or more.

Peritonitis Due to Chemic Irritants.—This may result from the bile irritating the serous surface. Toxins produced by bacteria in other organs may in many cases produce peritonitis. When resulting from chemic irritants, the inflammation is not of a purulent character, but fibrinous, and rarely hemorrhagic. Acute peritonitis may develop during the course of acute nephritis and arterial sclerosis. Disinfectants when applied to the peritoneum may produce the inflammation.

Peritonitis Due to Mechanical Causes.—Peritonitis may result from blows, or from wounds, such as stab wounds and gunshot injuries. When resulting from wounds, bacterial infection frequently follows.

Pathology.—Purulent peritonitis is always of bacterial origin. Without bacteria, pus does not form in the peritoneum. The reverse, however, is not true—that is, not every peritonitis due to bacteria must be purulent or (perhaps more correctly) go on to pus formation.

Chemic peritonitis is serous, serohemorrhagic, or fibrinous. The purely mechanical peritonitis is adhesive (fibrinous) only (Nothnagel). In opening the abdomen in cases of acute general peritonitis, the visceral and parietal layers will be found adherent as a result of the exudation; indeed, in some cases the matting together of the intestines is so marked that it is impossible to separate some of the coils of intestine from each other. The exudation varies from that of a serofibrinous character to fibrinous, fibrinopurulent, purulent, and in some cases hemorrhagic. The intestines are found distended in the majority of cases. When the exudation is purulent, the pus which is found in the peritoneal cavity may be thick and of a greenish color. In some instances some fluid or pus may be found in the dependent parts of the abdomen, particularly in the pelvis. The intestines are injected and often reveal marked inflammatory changes, so that the wall becomes very friable. They may often be gangrenous. Microscopically, the first change noted in acute peritonitis is the swelling and opacity of the endothelial cells, the blood-vessels becoming dilated and tortuous, and the exudate soon finding its way upon the free surface of the peritoneum; this exudate may be of one of the varieties just enumerated, the character depending upon the etiologic factor. In acute general peritonitis the disease is usually most marked nearest the original focus of inflammation.

Symptoms.—The symptoms may be ushered in by chilly sensations, or a distinct rigor with abdominal pain may mark

the onset. The pain may at first be local, but soon becomes general, increased by movement of the patient or by pressure upon the abdomen. The position of the patient is frequently characteristic. The thighs are partly flexed, so as to relieve the tension of the abdominal muscles, and the shoulders are elevated. Movements of the patient are restrained, as they produce intense pain; even the pressure of the bedclothes or paroxysms of coughing cause distress. Respiratory movements are restricted to the thorax (thoracic breathing). The pulse becomes rapid, small, and hard; it has been called the "wiry pulse." The rapidity of the pulse varies from 110 to 160 per minute, or may be even higher. Soon after the onset of the disease the temperature rises to 103° F. or 104°F., or higher, and continues throughout the attack. Hiccup is a distressing symptom, causing severe abdominal pain, and frequently precedes the fatal issue. The symptoms are frequently ushered in by vomiting, which is an important symptom of the disease. Efforts at vomiting cause intense abdominal pain. The vomited material is first that of the contents of the stomach, later fecal in character, owing to the regurgitation of the intestinal contents. Diarrhea may be an early symptom, but constipation soon follows; even flatus rarely escapes, the abdomen soon becoming distended, and may be markedly tympanitic. In rare instances the distention is not pronounced. Micturition is frequent; in some instances retention may be prominent. The urine is scanty, high-colored, and contains an increased amount of indican. When the disease is well advanced, the patient presents a characteristic appearance. The vomiting is constant. The expression of the face is anxious. The features are pinched and the eyes sunken, and marked wasting is apparent. On examination of the abdomen distention is noted. On *palpation* it is rigid, board-like, and extremely tender to the touch. *Percussion* reveals tympany. The lower border of liver dullness is obliterated. Splenic dullness may be completely obscured. The heart may also be displaced upward. If considerable effusion into the peritoneal cavity is present, flatness may be noted upon percussion in the flanks. Early in the disease friction sounds may be noted on auscultation, but this is rarely found.

The course of the disease varies greatly, depending upon its nature and extent. Death usually results; it may occur within twenty-four or forty-eight hours, or not for eight or ten days. As the fatal issue approaches, the surface temperature falls, the

internal temperature, however, being high (pre-agonistic rise); the respirations become shallower, the pulse more rapid, and occasionally death may result from paralysis of the heart.

When general peritonitis arises from perforation, symptoms of collapse are first present, which come on rapidly, and gradually the symptoms just enumerated arise. This is an exceedingly fatal form of peritonitis.

Diagnosis.—The direct diagnosis depends upon the intense pain in the abdomen, which may be continuous, increased upon movement or pressure and in urination; diminished amount of urine, with difficulty in voiding; tympany of the abdomen, vomiting, singultus (hiccup), constipation, fever, with small, frequent pulse, increased respiration of the thoracic type, the characteristic facies, and the symptoms of collapse. These symptoms are confirmed by the physical examination in finding the inflammatory exudate in the peritoneal cavity.

ACUTE LOCAL PERITONITIS.

The inflammation may be localized in various parts of the peritoneum, the most frequent situations being in the neighborhood of the appendix and in the pelvis. When in the latter position, there is usually inflammation of the sexual apparatus. It may involve the diaphragm, when it is called *diaphragmatic peritonitis*.

Diagnosis.—The diagnosis of this condition can be made only when there is an association of pain, exudation, and the friction sound in particular localities in the abdomen. The diagnosis should never be made from pain alone, as in the majority of instances mistakes will arise.

CHRONIC PERITONITIS (ADHESIVE PERITONITIS).

Etiology.—This may be *local* or *general*. It may result from repeated attacks of acute peritonitis, the causes having been enumerated in the etiology of the acute form. Occasionally chronic peritonitis may originate without being preceded by acute inflammation. Chronic peritonitis may result from tuberculosis or cancer; it follows trauma and operative procedures. Local chronic peritonitis is more common than the general form.

Pathology.—On examination of the peritoneum it may be found that great thickening is present, and fibrous adhesions may be noted. Effusion into the peritoneal cavity is sometimes observed.

Symptoms.—The symptoms are not characteristic, and the true nature of the affection is revealed only at autopsy in many instances.

Prognosis.—The prognosis of chronic peritonitis is usually favorable, but death may result from complications, such as intestinal obstruction or pressure upon the abdominal organs.

Treatment of Peritonitis.—Acute Peritonitis.—Absolute rest in the recumbent posture is necessary. A pillow may be placed under the knees so as to relieve the abdominal tension. A cradle may be placed over the abdomen in order to support the bedclothes. Food should be withheld while vomiting persists, and during this time rectal feeding is often of great value. When vomiting subsides, food should at first be given in small amounts, and only in the liquid form. Thirst should be relieved by supplying fluids by rectal injections. Small pellets of ice may be given the patient to relieve the dry condition of the mucous membrane of the mouth. After the stomach becomes retentive, water should be given in small amounts. Carbonated waters are frequently more palatable. Locally, cold is of great value in the treatment. A number of ice-bags should be placed upon the abdomen, these being retained as long as there are any signs of acute inflammation. Turpentine stupes may sometimes prove beneficial. In the early stages, when vomiting begins, this may be relieved by placing a small fly blister in the epigastric region just below the ensiform cartilage. Opium given in liberal amounts is of great value in the treatment of peritonitis. Early in the disease, when the diagnosis is still in doubt, as peritonitis is so often due to causes which necessitate surgical intervention, it is sometimes advisable to withhold the opium for a short time, as it will mask the symptoms; but if the diagnosis has been made, or if diagnosis seems impossible, it is necessary to administer opium freely. When vomiting ceases, calomel or salines should be administered. Various forms of enemata are of great value. Glycerin suppositories, asafetida suppositories, or the rectal tube may also be employed. Of late, operative measures consist in opening the peritoneal cavity, with or without drainage, this being recommended by some authorities. The operative treatment has been quite successful in suppurative peritonitis, or in the early stages, especially when due to intestinal perforation, intestinal ob-

struction, or appendicitis; therefore a surgeon should always be consulted when dealing with these cases.

The treatment of chronic peritonitis consists in maintaining the general health and in the administration of calomel and salines. The pain should be relieved by opium; great care must, however, be taken to avoid establishing the opium habit. If large effusions are present, they may be relieved by tapping. In chronic peritonitis the surgeon should be called without delay. Operative measures often serve to relieve the condition completely.

ASCITES.

Definition.—By ascites is meant an abnormal collection of fluid in the peritoneal sac.

Etiology.—The most frequent cause is obstruction of the portal circulation, as occurs particularly in atrophic cirrhosis of the liver. This obstruction may also be produced by other diseases of the liver, also by tumors or inflammatory masses pressing upon the portal vein. It may result from valvular heart disease; ascites, however, occurs late in the course of heart disease. Rarely emphysema, chronic pleurisy, and interstitial pneumonia may give rise to the condition. Bright's disease, and the cachectic states, particularly grave anemias, give rise to the condition. The affection may result from local disease of the peritoneum, such as cancer and tuberculosis. In rare instances a fatty, milk-like, chylous or chylous-like fluid is found in the peritoneal sac, which is due to the admixture of fat. Rarely is a chylous ascites encountered.

Symptoms.—The symptoms will depend upon the amount of fluid present in the peritoneal cavity, and upon the cause. When large quantities of fluid are present in the abdomen, the contour of the belly wall is changed; there is bulging at the flanks, and a depressed top. Occasionally, striæ may be noted upon the skin. The superficial veins are filled. Edema and engorgement of the veins of the lower extremities are sometimes encountered, these symptoms being due to the restricted circulation, as a result of the pressure upon the inferior vena cava which is produced by the fluid.

Upon combined palpation and percussion, a wave is elicited. This is best performed by tapping the abdomen on one side and palpating on the other, and it is well to have an assistant place his hand in the median line in a vertical position, so as to

avoid a wave being transmitted through the superficial parts. Upon percussion a tympanitic note is obtained in the upper part of the abdomen, particularly around the umbilicus, when the patient is in the recumbent posture, and a flat note will be heard in the flanks. On a change of posture the percussion note varies; in the sitting posture tympany will be elicited in the epigastrium, and dullness beneath. When there are adhesions of the intestines, binding these organs together, the change in the note does not always occur. It will also be noted that the liver, the heart, and the spleen are usually displaced.

Differential Diagnosis.—Ovarian Cysts.—Enlargement of the abdomen is produced by ovarian cyst, which is often enormous. It can be differentiated from the ascites in that the shape of the abdomen is different, the enlargement arising from the pelvis and extending toward the umbilicus or epigastrium, the upper portion being somewhat rounded, not flattened, as is the case in ascites; nor does the abdomen sag in the flanks in this condition. Fluctuation may be elicited upon palpation. Upon percussion, tympany is observed in the flanks, and dullness in the upper portion of the abdomen. The percussion note does not vary greatly when the posture of the patient is changed.

Prognosis.—The prognosis depends upon the cause. As a rule, it is unfavorable.

Treatment.—The treatment should at first be directed to the cause. When the fluid becomes excessive, tapping is necessary (*paracentesis abdominis*).

For the purpose of tapping, a trocar and a cannula are used. In performing this operation, care must always be taken that the bladder be thoroughly emptied. The point of selection is in the median line, midway between the umbilicus and the symphysis pubis. A many-tailed bandage may be used, traction being made upon the ends as the fluid flows through the trocar. The patient should be in the sitting posture. All the fluid may be withdrawn unless symptoms of collapse manifest themselves, when the operation must be suspended at once. When fluid reforms, which it does very rapidly in the majority of instances, another tapping is necessary. Occasionally the rapid formation of the fluid may be somewhat delayed by a tight bandage over the abdomen.

TUMORS OF THE PERITONEUM.

BENIGN TUMORS.

Fibromata are occasionally met with; they rarely attain great size, and seldom give rise to symptoms. Lipoma and fibrolipoma may attain enormous size. In a case recorded by Waldeyer, the weight of the growth was 31 ½ kilograms. These tumors may be single or multiple, and are most frequently found in persons between the ages of thirty and fifty. Angiomata are sometimes encountered. Myoma and fibromyoma have been encountered, but they are of rare occurence.

Symptoms.—The symptoms are due to the pressure produced by these tumors.

MALIGNANT TUMORS.

Sarcoma of the Peritoneum.—In this group are included those growths which spring from the peritoneal or the subperitoneal tissue, the most common situations being in the connective tissue behind the peritoneum, in the greater omentum, and in the folds of the broad ligament or upon the surface of the liver. The retroperitoneal lymphatic glands are also sometimes the site of origin.

Sarcomata of the peritoneum may be primary or secondary. When it is secondary, the primary seat frequently is in the testes or ovary, or sometimes in the kidney. They are found more commonly in males than in females, in adult life, sometimes at an early age, and there may be a history of trauma.

The varieties of sarcomata met with are the round-cell, spindle-cell, lymphosarcoma, and mixed-cell. They may be combined with fibromata, and in some instances reveal extensive myxomatous degeneration, and often melanotic pigmentation.

These tumors may be single or multiple, and attain an enormous weight—thirty pounds or more. Like all sarcomata, they are usually very vascular. They may be soft or hard, depending upon the fibrous elements which are so frequently combined.

Chronic inflammatory changes of the peritoneum are frequently associated. Interference with the normal lymphatic transudates in the peritoneum is sometimes noted, giving rise to ascites. From the pressure of these tumors or from the pressure of the ascites upon the vena cava, edema of the lower

limbs may be encountered. Interference with the portal circulation gives rise to distention of the superficial veins (collateral circulation).

Symptoms.—The symptoms greatly depend upon the size and situation of the tumor, also upon the fact whether it be primary or secondary. The patient often complains of vague abdominal pain, and sometimes of nausea, vomiting, and loss of appetite; there are loss of weight, failing strength, and general ill health.

Upon examination, the *wasting and anemia are usually distinctive signs.* Examination of the abdomen will usually reveal the true nature of the condition. If it be a sarcoma of the omentum, upon palpation a hard, irregular, flattened mass, tender upon pressure, will be noted. The tumor is freely movable from side to side, and light percussion will elicit a dull note. If the tumor be situated in the mesentery, it is usually palpable above or below the umbilicus, in the median line, is freely movable unless adhesions have taken place, and its shape is commonly globular. If the sarcoma be situated behind the peritoneum, and is of considerable size, it displaces many of the abdominal viscera, such as the pancreas, the intestines, and the kidneys.

Prognosis.—The course of these tumors is usually brief—a few months or a year, the prognosis in all instances being invariably grave.

Carcinoma of the Peritoneum.—Whether primary carcinoma of the peritoneum ever exists is doubted by some observers, and it seems probable that many of these tumors that have been described really belong to the class of alveolar sarcomata. The pathologic distinction between alveolar sarcoma and carcinoma is extremely difficult, and in some cases impossible.

Secondary carcinomata of the peritoneum are not infrequent, the primary seat commonly being the ovary, or less often the gall-bladder, pancreas, liver, stomach, intestines, esophagus, and breast. They are probably more common in women, occurring in middle or more advanced life, and occasionally in the young. When the peritoneum is involved secondarily, there will be observed a great number of nodules, the size of which varies from that of a pinhead to a pea or marble; they are often clustered. These miliary or multiple forms may be of a primary or secondary nature.

The tumor may be of comparatively large size, be quite

hard, and give the characteristics of scirrhous carcinoma. Occasionally large tumors which have undergone colloid degeneration have been recorded. There is often some degree of ascites, particularly when the carcinoma is multiple. The fluid contained in the peritoneal cavity may be serous, occasionally hemorrhagic, and rarely chylous.

Symptoms.—The onset is very insidious, the patient complaining of loss of strength, loss of appetite, and loss of weight. *Anemia and cachexia develop.* The symptoms are much influenced by the position of the tumor and its primary seat. Abdominal pain may be colicky in character, and is a very frequent symptom ; however, it does not occur in all cases. The temperature is usually subnormal. Constipation is the rule, but attacks of diarrhea often alternate, and vomiting may also occur. The loss of weight is, in some cases, enormous.

Upon examination of the abdomen, the physical signs of ascites, the palpation of the nodular mass, and pain upon pressure frequently reveal the true nature of the condition. When the tumors are large, they are usually movable, but late in their course they become bound down by adhesions.

Prognosis.—The disease is of short duration, rarely lasting longer than six months, and invariably terminating fatally.

Treatment of Malignant Tumors of the Peritoneum.—If the diagnosis be made early, while the tumor is small, operative measures may be of value. As a rule, however, complete removal is impossible. If ascites be pronounced, paracentesis abdominis may be performed. The diet should be nutritious and easily assimilable. The bowels should be regulated, and pain must be relieved by the administration of opium.

CYSTS OF THE PERITONEUM.

The peritoneal and subperitoneal tissue may be the seat of hydatid cysts, these frequently being multiple, also dermoid cysts, and rarely chylous cysts.

The signs and symptoms depend upon the location and the size of the cyst. Their duration is variable, and when they are of only moderate size they are frequently not detected until the autopsy is made. If the cyst be situated so as to cause some degree of intestinal obstruction, the symptoms relating to obstruction will occur ; if situated in relation to the common bile-duct, symptoms of jaundice will arise. The diagnosis is difficult in many instances.

DISEASES OF THE LIVER.

DISPLACEMENTS OF THE LIVER.

Displacements of the liver may be due to pressure from above the diaphragm, such as thoracic tumors, pleurisy with effusion, and effusion into the pericardium; to tumors or abscesses between the liver and the diaphragm; or to a relaxation of the hepatic ligaments. Of the latter, the most important causes are pregnancy at full term, a pendulous abdomen, stretching of the ligaments, and a tearing or twisting of the suspensory ligament. Tight lacing produces a change in the form of the liver rather than in its position, but both conditions may occur in the same case.

Symptoms.—A sudden onset is rare; when this occurs, there is pain in the hepatic region, with irregular, rapid pulse, a feeling of weight, and severe dyspeptic symptoms. There may be jaundice. In the greater number of cases the onset is gradual. The physical signs show the presence of a tumor in the abdomen, with the shape, size, and consistency of the liver. A point of importance in the diagnosis consists in the difficulty of replacing the liver in its normal position. If the liver be greatly displaced, a tympanitic resonance instead of flatness occurs in the normal hepatic area.

Diagnosis.—Floating liver may be mistaken for ovarian cyst, movable right kidney with hydronephrosis, and malignant disease of the omentum. Important points in the diagnosis are the upper smooth surface, with the sharply defined anterior border and notch, the tympanitic resonance over the hepatic region where normally liver flatness should exist, and the ability to partially or completely restore the displaced liver to its normal position.

Treatment.—Support of the abdominal walls to hold the liver in its normal position should be tried. Surgical treatment has been successful in several instances.

ACTIVE CONGESTION OF THE LIVER.

Definition.—An increased amount of blood in the liver due to an increase in the flow of blood through the organ.

Synonyms.—Hyperemia; active hyperemia.

Etiology.—The condition is physiologic during digestion. It occurs during active exercise, and results from overeating, overindulgence in malt liquors, wines, etc., and from the ingestion of highly seasoned foods. It results from malaria, dysentery, yellow fever, Weil's disease, and enteric fever. Cold is regarded as an etiologic factor. It occurs during the course of diabetes mellitus, and sometimes from suppressed menstruation.

Pathology.—The pathologic changes on the postmortem table are not constant, and are often ill defined. The liver may be enlarged and of a dark red color, and on section blood drips from the cut surface. On viewing the cut surface it may be slightly mottled; this, however, is never so pronounced as in passive congestion (the "nutmeg" liver), in the latter condition it being due to the distention of the intralobular veins. On microscopic examination cloudy swelling sometimes accompanies hyperemia.

Symptoms.—The symptoms are those of gastro-intestinal catarrh, such as constipation, loss of, or perverted appetite, coated tongue, and headache. The complexion of the patient is often muddy. There may be slight pain or a sense of discomfort in the region of the liver, and some tenderness on pressure. Slight jaundice sometimes arises; rarely is this intense. Mental depression, irritability, headache, and dizziness are often prominent symptoms.

Upon examination, the liver may be found slightly enlarged, but this is difficult to determine.

Prognosis.—The disease frequently terminates in more serious affections of the liver, it being in itself not dangerous.

Treatment.—Errors in diet must be corrected, and the food should be light, nutritious, and easily assimilable. Highly seasoned foods must be avoided. In many instances a milk diet may be instituted. Alcohol must be avoided. Systematic purgation is necessary. It is best to administer Carlsbad salts, Rochelle salts, and calomel. Pills of aloin, belladonna, and strychnin are frequently of use. Benefit may be derived from a visit to some watering-place, such as Carlsbad, Homburg, etc.

PASSIVE CONGESTION OF THE LIVER.

Definition.—A condition characterized by enlargement of the liver, and due to an interference with the outflowing blood from the organ, the cause in nearly all instances being heart disease.

Synonyms.—Passive hyperemia; congestion; nutmeg liver; cyanotic liver; red atrophy of the liver; liver of heart disease.

Etiology.—This depends upon the interference of the outflowing blood from the hepatic veins, so that the liver contains a greater amount of venous blood than normal. As before stated, in nearly all instances it is due to a cardiac lesion, particularly when the right heart fails to do its work, and the blood dams back into the inferior vena cava and the hepatic veins. The blood may be retarded as a result of pulmonary disease, such as chronic interstitial pneumonia, compression of the lung (atelectasis), pleural effusions, intrathoracic tumors, and aneurysms. In rare instances it may be due to pressure upon the hepatic veins or upon the inferior vena cava, causing a constriction.

Pathology.—The organ is enlarged, but this enlargement is never so great at the autopsy as it is antemortem, the blood tending to leak out of the organ after death. The surface is smooth and the edges are rounded, and it is of a bluish-black color. On section the knife meets with more resistance than normally, as a rule. The cut surface appears mottled, and drips blood. Upon microscopic examination it will be found that the intralobular veins are greatly distended with blood and dilated. As a result of this, many of the liver-cells surrounding these veins are atrophied, large numbers having disappeared. Yellowish-brown pigment is deposited in many of the surrounding liver-cells. Fatty infiltration is also observed in the portal vein zone—this being due to the slowing of the circulation. The mottled appearance is due to the engorged intralobular veins, the color being blue or black, and to the contrasting light color of the surrounding liver-cells, many being infiltrated with fat. If the condition be of long standing, fibrous connective-tissue formation is often noticed around the intralobular veins. Secondarily, the portal circulation may be disturbed, giving rise to enlargement of the vein and to ascites.

Symptoms.—These depend more upon the primary condition than those produced by the enlargement of the liver, the patient often complaining of a sensation of tension or pain in the right hypochondriac region.

Upon palpation, pulsation is sometimes noted, the liver also being found enlarged and tender to the touch. The gastro-intestinal symptoms depend upon the degree of portal congestion. Slight jaundice and the occurrence of ascites are also encountered.

Prognosis.—The prognosis depends entirely upon the cause of the congestion.

Treatment.—The treatment must be directed toward the cause; and if due to valvular heart disease, the use of digitalis frequently relieves the condition. Calomel and Rochelle and Carlsbad salts are of value in the treatment. If the local symptoms are severe, the application of leeches or blisters may be of some use.

FATTY LIVER.

Synonym.—Steatosis of the liver.

Etiology.—The distinction between fatty infiltration and fatty degeneration of the liver is by no means clear, and as yet too little is known of the changes that take place in fatty degeneration, or the reason of fat production in the liver cells. The normal amount of fat in the liver fluctuates; it is commonly between 3% and 5%. In fatty liver as much as 40% and over has been found. This condition may be physiologic, occurring after a full meal, particularly after partaking of food consisting largely of carbohydrates. It is physiologic in infants, being due to the milk diet. It results from the partaking of large quantities of carbonaceous food; from deficient oxidation, this being met with in tuberculosis of the lungs and certain of the anemias, as pernicious anemia and chlorosis. It arises from the excessive use of alcohol, from insufficient exercise, and from the slowing of the blood through the liver, as occurs at the onset of atrophic cirrhosis, or from passive congestion; and, finally, it occurs as a hereditary condition, obesity being common in many families. It may be due to poisoning, particularly by phosphorus; or it may result from poisoning by arsenic, antimonium, copper, mercury, and aluminium salts. It may also be due to the mineral acids, such as nitric, hydrochloric, and sulphuric acids. It has been observed after toxic doses of carbon dioxid, chloroform, iodoform, and carbolic acid, from the continued use of morphin, from poisonous mushrooms, and from the ptomains of flesh, fish, and mussels. It has been noted

after partaking of poisoned maize, as in *pellagra*. It occurs as a result of some of the infectious diseases, particularly yellow fever, puerperal fever, osteomyelitis, and erysipelas, and in conditions in which long-continued pus formation occurs. It has been noted after variola, severe diphtheria, and scarlet fever, and occasionally after typhus, cholera, and pneumonia. It rarely takes place in the course of chronic dysentery, and may occur in rachitic children.

Pathology.—In well-marked fatty infiltration the organ is increased in size, the surface is smooth, and the edges are somewhat rounded. The total weight is increased, but the specific gravity is decreased. The color is of a lighter shade than normal, being yellowish. On section, the knife meets with little resistance, and fat globules may appear upon the knife, the cut surface being of a yellowish-red color, and frequently mottled, there being areas of a light yellow color intermingled with those of a reddish shade. Sometimes large portions of the organ are of a light yellow color. Upon microscopic examination it will be found that the outer zone of the liver lobule particularly is invaded with fat; many of the liver-cells are very large, being distended with fat, and the nucleus being pushed to one side. If the infiltration be pronounced, other portions of the liver lobule will be involved. Pathologically, it is sometimes difficult or impossible to distinguish fatty infiltration from fatty degeneration. In well-defined cases of fatty degeneration the size of the organ is decreased; it is very friable, soft, and sometimes semifluid, and fluctuating. It is of a deep yellowish-red color, and upon microscopic examination it will be found that many of the liver-cells are filled with small globules of fat, others revealing parenchymatous degeneration.

Symptoms.—Upon palpation the liver is noted to be soft, and the inferior margin is found lower in the abdomen than normally. Upon percussion, it is observed that the organ is considerably enlarged. The surface seems smooth, the margin rounded, and the general outline of the liver remains the same as normal. Ascites and enlargement of the spleen are rare; if present, they are due to some complication. Gastro-intestinal disturbances are common; there is loss of appetite, flatulency, and constipation alternating with diarrhea, the stools containing mucus. Meteorism is common. Hemorrhages are frequently noted. Jaundice is rare. The bile is diminished in amount due to the decreased activity of

the liver-cells. Upon examination of the urine, it will be noted that urobilin is excreted in diminished amounts. The appearance of the patient and the general symptoms depend largely upon the cause.

Course and Duration.—The disease runs a chronic course, being much influenced by complications, which are frequently present, such as fatty heart and fatty kidney.

Treatment.—If possible, efforts should be made to remove the cause. If this be obesity, fatty and saccharine food should be eliminated from the diet, and albuminoid substances substituted. Meat, fish, green vegetables, and plain broths may be given. All stimulants should be avoided, especially beer and sweet wines. Systematic exercise should be insisted upon. The alkaline waters, Carlsbad, Vichy, and Kissingen, are beneficial. If there be anemia, iron and arsenic are of use.

AMYLOID DISEASE OF THE LIVER.

This is part of a general process, in which the spleen, kidneys, and intestines are also frequently involved. Occasionally it results from hereditary or acquired syphilis. The disease may be congenital, and is found at all ages from two to seventy years, occurring most frequently between the ages of twenty and thirty. It is more frequently found in men than in women. The disease is often due to suppurative processes, especially those affecting bone, also chronic malaria and tuberculosis.

Pathology.—The organ is greatly increased in size and weight. The surface is quite smooth and the edges are rounded. The specific gravity is increased. The color of the organ is a pale yellow. On section the knife meets with much resistance. The organ is tough, elastic, and pits but slightly upon pressure.

Test for Amyloid Material.—When a small section of the liver is treated with a weak iodin solution (or Lugol's solution), the amyloid material will stain a mahogany brown, and the remaining liver substance a light yellow. The test is also well demonstrated with some of the anilin dyes, such as gentian-violet, a rose-red color being developed when a solution of this kind is applied. Similar stains, such as methyl-green and methyl-violet, give like reactions.

Microscopic examination will reveal the amyloid material

infiltrated into the liver lobule. Early in the disease the hepatic artery or middle zone is first involved; later in the course of the affection the cells in the inner and outer zones are also involved. The smaller arteries also reveal amyloid material in their coats.

Symptoms.—The liver is found enlarged if there be an extensive deposit of amyloid material; occasionally it is found smaller than normal, the surface being smooth and firm, and the lower margin rounded and distinctly palpable. Pain is present if perihepatitis occurs. Ascites is rare. Enlargement of the spleen results in more than one-half of the cases. Unless some complication occurs, such as an enlargement of the lymphatic glands in the portal fissure, jaundice is absent. Dyspeptic symptoms are common; there is anorexia, nausea, and vomiting. Diarrhea, and anemia with leukocytosis, are common symptoms. The appearance of the face is characteristic; there is sallowness with pallor, the patient suffering from more or less debility. If edema be present, it is most likely due to a similar change in the kidneys.

Course and Duration.—The disease often extends over many months; the course, however, is progressive, death resulting from anemia, kidney complication, or some intercurrent affection, as pneumonia or dysentery.

The prognosis is always unfavorable. If the disease be due to suppurative processes, these will require careful attention.

Treatment.—The general hygiene of the patient should be carefully looked after. The diet should consist of nutritious food, containing very little fatty, farinaceous, or saccharine material. If the disease be of syphilitic origin, iodid of potassium and mercury are of use.

ATROPHIC CIRRHOSIS OF THE LIVER.

Synonyms.—Chronic interstitial hepatitis; hobnail liver; gin-drinker's liver; finely granular liver; Laennec's cirrhosis.

Etiology.—The disease occurs particularly in the male sex, and is often due to alcohol. In the new-born the disease is due to hereditary syphilis. The second most important cause is acquired syphilis, malaria, and other infectious diseases, as cholera, enteric fever, and scarlet fever. Gout and rickets particularly give rise to this condition. The disease has been known to follow miliary tuberculosis. It also arises in the course of perihepatitis. It may accompany red atrophy of

the liver. The individual predisposition is of some importance as an etiologic factor. Occasionally the disease occurs without assignable cause.

Pathology.—The liver is commonly greatly diminished in size and in weight, but the specific gravity is increased. The surface is irregular, being lobulated or finely granular; the edges, also, are nodulated. On palpation it is found to be extremely tough—india-rubber-like. On section, the knife meets with much more resistance than the normal organ. The color is lighter than normal, being yellowish-red, or in some instances a light red. Some of the lighter areas indicate bands of fibrous connective tissue which traverse the organ.

Microscopic examination reveals a great increase in the connective tissue, which is especially well marked around the lobules and interlobular vessels. As a result of the contraction of this new cicatricial tissue, many of the liver-cells disappear from pressure atrophy, and the finer blood-vessels are pressed upon.

The radicles of the portal vein, because of their weak walls and of the low blood pressure in the vessels, seem to suffer more than the accompanying vessels. For this reason portal obstruction results. The cicatricial bands are found traversing the liver, many of the lobules being distinctly defined. Large numbers of these bands reach to the surface, and, on account of the secondary contraction, produce a distortion in the outline of the liver (hobnail liver). The portal obstruction leads to congestion of the spleen, this organ becoming large and of a dark red color. It also leads to congestion of the stomach, the small intestines, and the large intestine, this being especially marked in the rectum. The hemorrhoidal plexus of veins becomes distended, tortuous, and elongated, giving rise to hemorrhoids. The portal congestion causes transudation of the serum into the peritoneal cavity, and ascites results. Collateral circulation, that connecting the portal with the general venous system, is in most instances established, so that the superficial abdominal veins become greatly distended. The left ventricle of the heart may show slight hypertrophy. In the early stages of cirrhosis the liver usually presents slight enlargement and some degree of fatty infiltration, the latter probably resulting from slowing of the circulation. Very late in atrophic cirrhosis of the liver the contraction of the connective tissue may be extreme, giving rise to narrowing of the biliary ducts.

Symptoms.—The prodromal stage may occur without giving rise to symptoms, except in the case of topers in whom there is a long preceding history of gastro-intestinal catarrh. There are symptoms of anorexia, sensations of pressure in the epigastrium, constipation alternating with diarrhea, early morning nausea and vomiting, and so on. Early in the course of the affection some slight enlargement of the liver may sometimes be made out by the methods of physical diagnosis. When the disease has established itself, the organ becomes distinctly diminished in size. Occasionally the diminution in the size of the liver is very difficult to determine, owing to the fact that shrinkage occurs in the diaphragmatic portion of the liver. Of decided diagnostic import is an early atrophic change in the left lobe of the liver, which in some cases can scarcely be felt in the abdominal cavity. If perihepatitis occur, distinct fremitus may be elicited in some cases upon palpation. Jaundice, as a rule, is absent; and if present, it is slight in amount, occurring only late in the course of the affection. The symptoms depend upon the disturbance in the circulation of the blood in the portal vein, or upon the diminished function in the atrophied cells of the liver, or upon a combination of these two affections. The most characteristic symptoms are those relating to disturbance of the circulation in the portal vein. The result of this is a marked ascites. Even before this the effect of congestion of the mucous membranes of the stomach and bowels may be noted by hematemesis and enterorrhagia, which occur in the course of this affection. With this there may be constipation, alternating with diarrhea; and meteorism is often a prominent symptom. Soon hemorrhoids begin to show themselves, as a result of the overfilled portal circulation. The spleen is enlarged. The ascites is often pronounced, and may be so great as to push the diaphragm so far upward as to compress the lungs and heart, giving rise to dyspnea, and often orthopnea. The pulse is irregular and intermittent, from the disturbance of the heart.

From the pressure occasioned by a large effusion, interference with the venous return in the inferior vena cava is produced, this giving rise to edema of the lower extremities. The cutaneous veins of the abdomen are often greatly enlarged; this frequently shows itself around and in the neighborhood of the umbilicus, giving rise to the so-called "caput medusæ." The urine is diminished in amount, of low specific gravity, and reddish in color. If albuminuria occur, it is due to a complicat-

ing urinary affection, the same sclerotic process very often going on in the kidney. The red color of the urine is due to the urobilin, which is often excreted in increased amounts. Urea, as a rule, is diminished. Occasionally, traces of sugar may be found. The occurrence of sugar in the urine of persons suffering from atrophic cirrhosis may be explained by a disturbed metabolism in the organ. Occasionally leucin and tyrosin have been found (?).

The temperature, as a rule, is normal or subnormal, subfebrile or febrile ranges being due to complications. The general nutrition suffers greatly. The patient becomes thin, the muscle tonus is lost or diminished, and there is marked anemia. In some cases cerebral symptoms occur, with delirium, stupor, and coma. Rarely convulsions occur. These are said not to be due to *uremia* or *cholemia*.

Complications.—The most common complications are interstitial and parenchymatous nephritis. Myocarditis also occurs. Complications which relate to the liver are found in fatty infiltration and passive congestion. Abscesses, amyloid disease, and carcinoma occasionally develop in the cirrhotic liver. Sometimes large hemorrhages may occur from the lungs, from the urinary passages, and from the nose (epistaxis). Hemorrhages from the stomach and bowels have already been mentioned among the symptoms; these may be profuse, and are the most frequent forms of hemorrhage. Epistaxis has occasionally led to the fatal issue.

Prognosis.—The course of the disease is prolonged, much depending upon the compensatory collateral circulation, the duration in many cases being ten years or more; however, always terminating fatally. The cases due to malaria and syphilis are more favorable if the condition be diagnosticated early.

Treatment.—The treatment consists in abstaining from alcohol, avoiding rich foods, and leading a quiet life. The bowels should be carefully regulated, and the skin kept in a good condition by frequent bathing. Iodid of potassium and mercury are of use only in the syphilitic forms of the disease. When the disease is well advanced, the bowels should be kept freely open by the use of calomel, salines, compound jalap powder, elaterium, etc. When the ascites is pronounced and pressure develops, paracentesis abdominis becomes necessary. The double chlorid of gold and sodium is recommended in the beginning of the disease.

BILIARY CIRRHOSIS.

Definition.—Cirrhosis due to obstruction of the gall-ducts, bile being retained in the liver.

The French school of physicians differentiated two forms of cirrhosis—a hypertrophied biliary cirrhosis (Hanot), due to disease of the small biliary passages, and a biliary cirrhosis due to obstruction, with sclerotic change in and around the larger gall-ducts, producing thickening of these ducts.

Pathology.—The organ is large and tough, and upon section presents a reddish-yellow color, sometimes "nutmeg" in appearance, this being due to the yellow discoloration of the central part of the liver lobule surrounded by the red periphery. The surface of the organ is quite smooth, and rarely becomes granular. The biliary ducts are dilated, and present sclerotic thickening around them. The gall-bladder may also reveal thickening and some catarrhal inflammation. This change may also exist in the large ducts. The newly formed connective tissue which exists around the lobules may push its way into the lobules.

Symptoms.—Jaundice coming on rapidly, with perhaps the symptoms of hepatic colic, is often characteristic. The jaundice in some cases may disappear after the passage of the stone. If the liver remains enlarged, there is great similarity between this and the ordinary form of cirrhosis; however, in this disease the jaundice is more marked, and ascites occurs.

Treatment.—The treatment is expectant symptomatic.

CONGESTION CIRRHOSIS.

In the majority of cases red atrophy is not accompanied by marked cirrhotic change, but in a few instances in which congestion is long continued new-formed fibrous connective tissue develops, giving rise to cirrhosis. The organ will be found hard, and the surface slightly granular. The capsule is wrinkled, and the organ is reduced in size. On section it presents the characteristic appearance of red atrophy, showing the dark red central zone of the lobule and the lighter peripheral portion. Upon microscopic examination the characteristics of red atrophy with new-formed connective tissue are observed.

Symptoms.—The symptoms are those of atrophic cirrhosis, usually complicated by valvular heart disease.

CIRRHOSIS DUE TO MALARIA.

This form of cirrhosis is rare. Osler says that in a large number of malarial cases observed in the Johns Hopkins Hospital during the last nine years not a single case of cirrhosis complicating this disease was noted. It is said that the melanin which lodges in the liver produces the chronic inflammation, fibrous connective tissue developing in large amounts. Jaundice accompanies the cirrhotic condition. Iron pigmentation is noticed in the liver-cells nearest the central and peripheral zones. In this form of cirrhosis there are pain upon pressure, vomiting of bile, and biliary diarrhea, which is often due to biliary pigments and hemoglobin. This occurs particularly in the *black water fevers*, and in other forms of *tropical malaria*. This form of cirrhosis is particularly well marked in malarial cachexia, the liver being extremely large, weighing from 2000 to 3000 grams, revealing *perihepatitis* and marked pigmentation.

SYPHILITIC CIRRHOSIS.

This may be due either to congenital or acquired syphilis. The liver becomes enlarged, tough, and resistant, being as hard as sole-leather, in a measure resembling amyloid liver.

Upon microscopic examination it will be observed that there is a great increase in the connective tissue between the lobules, many areas revealing numerous round and spindle cells. Gummata may also be found in this condition. These may vary from the size of a millet-seed to that of a walnut, and sometimes even larger. These gummata vary from a reddish-green to a creamy white color. They are often surrounded by a zone of fibrous connective tissue, and when contraction of this newly formed tissue occurs, marked distortion of the organ results. The gummata may occur in the liver without extensive cirrhosis during the course of acquired or congenital syphilis.

Symptoms.—The symptoms are those of atrophic cirrhosis—ascites, loss of weight, gastric derangements, anemia, and, late in the course, slight jaundice.

Diagnosis.—The diagnosis depends upon the history of infection, with enlargement of the organ.

Treatment.—Antisyphilitic treatment should be instituted early.

HYPERTROPHIC CIRRHOSIS.

Synonyms.—Hanot's cirrhosis; biliary cirrhosis; enlarged cirrhotic liver.

Etiology.—This is a comparatively rare affection, occurring in the male sex, most frequently between the ages of twenty and thirty-five. Very little is known about the etiology. It has been said that malaria, syphilis, enteric fever, and cholera are predisposing factors. Alcohol has also been mentioned as a predisposing factor. The disease appears to be much more frequent in France than in other parts of the world. Lately the hypothesis has been advocated that it is due to a primary parasitic disease of the biliary passages. It is possible that protozoa and bacteria may have some share in the process.

Pathology.—The organ is greatly increased in size, in some instances weighing as much as 4000 grams. The surface is granular. The portion of the peritoneal coat which lines the liver is frequently adherent to the organ, and is thickened. The liver is tough, cuts with much resistance, and is bile-stained, giving the organ a yellowish-green color. Microscopic examination reveals large masses of fibrous connective tissue between the lobules, and it is said that this connective tissue does not show the marked tendency to contraction that is so characteristic of the atrophic form. Aufrecht called attention to the fact that all the liver-cells are enlarged, and contain more than one nucleus. In the interlobular portions of the organ large numbers of round cells are frequently noted, as well as a number of fibroblasts and some fully developed connective tissue. The biliary passages show catarrhal change, and there is also a great increase in the number of ducts. The spleen is greatly enlarged, and the various tissues of the body are usually deeply bile-stained. Leukocytosis may be present.

Symptoms.—The early symptoms of the disease are not characteristic. They may consist of irregular gastric phenomena, such as nausea, loss of appetite, and a sensation of pressure in the epigastrium. It is only when the liver enlarges and becomes painful, and jaundice develops, that the symptoms of the disease become characteristic. At the height of the affection the liver is greatly enlarged, and may encroach upon the normal thoracic area. The liver is tender upon palpation. The jaundice is pronounced. The spleen is greatly enlarged, being easily determined by palpa-

tion. If pain occurs in the splenic area, it is due to a perisplenitis. Ascites does not occur. If fluid be found in the peritoneal cavity, its presence is due to complicating peritonitis. At some time in the course of the disease the appetite, which is at first lost, returns; bulimia may even be a symptom. The general nutrition of the patient, however, suffers considerably, and he rapidly loses flesh and strength. The urine is diminished in amount, is concentrated, and of a high specific gravity, containing bile pigment. Polyuria is sometimes associated with marked improvement in the condition of the patient.

The course of the disease is protracted. From the onset of the icteroid symptoms the disease may last from four to twelve years. In the later stages of the disease the jaundice becomes more marked, hemorrhages develop, and an intermittent fever shows itself, death being due to asthenia or to complications. It occasionally happens that toward the close of the affection the liver begins to shrink in size. Arthropathies affecting the fingers and toes, and even some of the larger bones of the extremities, have been noted in the later stages of the disease.

Complications.—These consist in peritonitis, myocarditis, and such changes in the heart as dilatation and hypertrophy. Anemia occasionally occurs, and there is some slight degree of leukocytosis, from 9000 to 20,000 per cubic millimeter. Urinary disease and albuminuria are rarer than in atrophic cirrhosis. Occasionally in the later stages of the disease the hemorrhagic diathesis develops. There may be epistaxis, hemorrhages into the skin, from the gums, and from the intestinal tract.

Diagnosis.—

	Atrophic Cirrhosis.	*Hypertrophic Cirrhosis.*
Liver	Small, usually granular.	Markedly increased in size; granular element not well marked.
Jaundice	Absent, as a rule; when present, not well marked.	Always present and well marked.
Ascites	Marked.	Absent; occasionally occurs toward the close of the disease, and often then not well marked.
Spleen	Increased in size.	Markedly increased in size.
Hemorrhages . .	Principally from the stomach and bowel.	Also marked from other parts.
Onset	Insidious.	With recurring attacks of gastric disturbance.
Duration	From two to three years.	From five to ten years.

	Atrophic Cirrhosis.	*Hypertrophic Cirrhosis.*
Complications .	Chronic contracted kidney and tubercular peritonitis comparatively frequent.	Very rare.
Age	After the fortieth year.	Usually before the fortieth year.

Prognosis.—The prognosis is always unfavorable.

Treatment.—The treatment consists in directing attention to the catarrhal condition of the stomach and intestinal tract. The diet should be a bland, unirritating one, and alcohol should be avoided. Iodid of potassium and calomel in continuous small doses have been highly recommended. Arsenic occasionally is of use.

ACUTE YELLOW ATROPHY OF THE LIVER.

Definition.—An acute disease of the liver characterized by severe nervous symptoms, vomiting, and hemorrhages, with an associated diminution in the size of the organ, due to parenchymatous and fatty changes.

Synonyms.—Acute parenchymatous hepatitis; icterus gravis.

Etiology.—This is an exceedingly rare disease, and there have been but 250 cases recorded in medical literature up to the year 1894. It most frequently occurs between the ages of twenty and thirty, no age, however, being exempt. One case has been recorded four days after birth. Females are more frequently affected. Pregnancy appears to play a predisposing part. Season is without influence. No relation has been traced between syphilis and acute yellow atrophy; the same is true of alcohol. Toxic elements appear to have a very close association, and the changes occurring in this condition resemble poisoning by phosphorus; in fact, many symptoms are common to both conditions. It has been claimed by some authorities that mental emotion appears to predispose; there is, however, no proof of this.

Pathology.—The organ is flabby, greatly reduced in size, weighing as little as from 90 to 120 grams, being so greatly reduced that on opening the abdomen it is hidden under the diaphragm. The surface is smooth, the capsule is wrinkled, and the color a yellow or dull red, the gall-bladder usually being empty. The consistency of the organ is quite firm, this being due to the fact that the connective tissue and the blood-

vessels are more or less well preserved, while the secreting or essential portion is atrophied.

Upon viewing the organ microscopically, a fine granular mass represents the greater portion of the hepatic cells. In many portions fatty degeneration has followed the cloudy swelling, and leucin and tyrosin crystals are often observed. The interstitial parts of the organ—that is, the fibrous connective tissue—and the blood-vessels are quite distinct. The spleen is commonly enlarged. It has been suggested that the disease is caused by a micro-organism.

Symptoms.—The disease begins as an ordinary attack of catarrhal jaundice. There is loss of appetite, nausea, vomiting, and epigastric distress, and this is followed in a day or two by the appearance of jaundice. One symptom is, however, of importance, and that is the occurrence of some rise in the temperature early in the course of the attack. This stage may last from five days to a week, but may vary considerably. The bowels are constipated; the tongue is coated, and the pulse ranges from 60 to 70 per minute. The usual signs of jaundice in the skin are apparent. As a rule, about this time a sudden change occurs in the clinical picture; there is marked, repeated, and severe vomiting, the patient rapidly becoming drowsy, semiconscious, and often delirious; the delirium may be maniacal, and the jaundice becomes intensified and of a greenish hue. The tongue is dry and brown; the pulse is rapid—from 120 to 140 per minute; and the respiration is quickened. The temperature falls, becoming subnormal. The vomiting has been almost continuous; the vomited matter now shows traces of blood. Enterorrhagia may occur, the stools being dark and offensive. There may be epistaxis and bleeding from the mouth, and petechiæ may occur. In women metrorrhagia, and in pregnant women abortion or premature birth, occurs. Associated with these symptoms, marked changes occur in the liver.

Upon *physical examination* it will be noted that dullness in the hepatic area is markedly diminished; in severe cases it may disappear altogether.

In the urine characteristic changes are noted, and the amount of urine is diminished. Leucin, tyrosin, and albumin are present. Bile pigments are increased in amount. The second stage is of extremely short duration, lasting only two or three days, the patient dying with symptoms of delirium, and in convulsions.

Duration.—The duration in the majority of cases is about fourteen days, rarely exceeding three weeks.

Prognosis.—The prognosis is absolutely unfavorable.

Treatment.—The treatment is symptomatic.

ABSCESS OF THE LIVER.

Etiology.—The cause of purulent inflammation of the liver is the entrance of pyogenic micro-organisms into the organ. The methods by which they gain access are various.

In abscess of the liver many organisms have been found associated, the most common being the streptococci, the staphylococci, the bacillus coli communis, Fränkel's pneumococcus, the bacillus typhosus, the bacillus pyocyaneus, the ray fungus, and, in the tropical variety, the amœba coli. The carriers of these micro-organisms are usually emboli, fish-bones, and parasites, such as worms.

Two varieties of liver abscess are differentiated — the *primary* and the *secondary*.

Those forms of abscess, originating from trauma, occurring from other organs from continuity of structure, as the gall-bladder or gall passages, are known as *primary* abscesses.

Secondary abscesses are those which are due to micro-organisms which have been carried into the organ by the blood stream. Etiologically, it is impossible to draw well-defined lines of distinction.

The most common are the secondary abscesses. These may occur from the morbid process being carried through the blood stream, which may be either through the hepatic artery or through the portal vein, and by some it is believed that it may even occur through the hepatic veins.

Through the hepatic artery the infectious process arises from pyemia, from ulcerative endocarditis, gangrene of the lungs, putrid bronchitis, etc.

When the mode of infection is through the portal vein, it is often due to appendicitis or ulcerative lesions of the bowel, or it may result from disease of the pelvic organs. This mode of infection most frequently arises in the course of tropical dysentery. Budd has claimed that an abscess in the liver in the course of dysentery is always of pyemic origin; however, the autopsies of such cases do not confirm this, as

there is most frequently only one large abscess, more rarely two. Abscess of the liver due to dysentery occurring in the tropics is very much more frequent in males than in females, and Europeans are much more likely to be affected than natives. This has been explained upon the basis that alcohol is a prominent predisposing cause, Europeans using more alcohol in hot climates than do the natives.

Pathology.—Multiple, Pyemic, or Embolic Abscesses of the Liver.—When the micro-organisms are carried to the liver through the portal vein, multiple abscesses arise in the organ, usually without abscesses in other parts of the body; but when the infection is conveyed to the organ through the arterial stream, abscesses are produced in many organs. From emboli the abscesses are rarely large or solitary. The liver containing pyemic abscesses is slightly enlarged, the surface usually being smooth and apparently normal. Upon section, a number of yellowish, rounded areas are exposed. These are filled with pus, the color of which varies, often being yellowish, grayish-green, or green. The edges of the abscess are irregular, and the cavities vary in size from a pin-point to masses about 6 cm. in diameter, rarely larger. The portal vein usually reveals suppurative pylephlebitis, and occasionally the biliary passages show a suppurative infection, which may extend to the gall-bladder. Upon microscopic examination the characteristics of acute suppuration are observed. Leukocytosis usually exists.

Pathology.—Large Solitary or Tropical Abscess.—These abscesses are usually very large. They may be solitary or, more rarely, multiple. The liver is greatly enlarged by the abscess; the organ may weigh 3500 grams. The abscess, as a rule, involves the right lobe. It frequently reaches a very large size, and in long-standing cases marked connective-tissue thickening occurs around it, so that it becomes hard and tough. The pus is usually thin and of a grayish or brownish-red color.

Upon microscopic examination the liver near the abscess reveals interstitial change. The abscess wall shows a dense fibrous connective tissue. The innermost portion of the wall is composed of a number of round cells, polynuclear leukocytes, and amebæ. Bacteriologic examination of the pus has demonstrated the fact that it may be sterile, or that it is infected with some of the micro-organisms of suppuration. The pus has been known to perforate into the peritoneal

cavity, the pleural cavity, the lungs, the colon, the hepatic and biliary vessels, and the inferior vena cava.

Symptoms.—Pyemic Abscess of the Liver.—The symptoms are those of pyemia, there being rigors, fever, and sweats, marked anemia and jaundice often being present. The liver is enlarged, and tender upon pressure; however, diagnosis of the abscesses is difficult or often impossible.

Tropical Abscess.—The liver becomes enlarged and tender, and jaundice, anemia, and wasting develop. The clinical manifestations vary somewhat, and large abscesses are occasionally present without marked disturbances; therefore, the symptoms may be divided into the *acute*, *subacute*, and *chronic* forms.

In the *acute variety* the constitutional symptoms are marked, there being extreme anemia, emaciation, marked rigors, fever, and sweating. There is pain in the region of the liver, which often radiates to the back and toward the right shoulder, or downward into the lumbar region. The liver is tender upon pressure, often giving rise to gastro-intestinal disturbances, painful respirations, and cough, while perihepatitis and peritonitis may exist with the suppurative inflammation of the liver and give rise to severe pain. The pain is often of a throbbing character.

Upon examination of the abdomen, enlargement is often noticed in the right hypochondriac region. Upon palpation a large, rounded, hard, and rarely fluctuating tumor is noted, and perhaps some edema of the abdominal wall. The chills, fever, and sweating occur with marked periodicity (daily); however, the patient may sink into the "typhoid state."

In the *subacute* and *chronic cases* the onset is more insidious and the symptoms are not so grave. There may often be an absence of fever. Weakness becomes extreme. Diarrhea may be present. In the acute cases the disease runs a short course of from fifteen to twenty days, the mortality being high. In the subacute and chronic forms the course is much longer, varying from four or five weeks to months.

Diagnosis.—When the disease is well advanced the diagnosis as a rule is easy, and depends upon the painful enlargement, which is sometimes fluctuating (if very superficial), the constitutional disturbances consisting of marked wasting, anemia, and the chills, fever, and sweating. The presence of the plasmodium in the blood is usually all that is required to differentiate it from malaria.

Prognosis.—The prognosis of pyemic abscess is extremely unfavorable, also that of tropical abscess, the mortality ranging from about 45% to 80%.

Treatment.—The treatment consists in the early evacuation of the abscess in suitable cases. The bowels should be regulated, being purged with calomel or salts. For the pain, opium in some form gives relief. When the septic phenomena are marked, free stimulation should be resorted to, whisky and strychnin giving good results. Quinin is often of use. For the anemia, iron and arsenic should be administered. The diet should be light and nutritious.

BENIGN TUMORS OF THE LIVER.

Fibromata.—These tumors are commonly so small that they do not give rise to distinctive symptoms; therefore the antemortem diagnosis is impossible. It is stated that these tumors not infrequently occur in the liver. They may be situated near the periphery or near the center. They exist in small masses, varying in size from a pinhead to a pea. On section, the knife meets with considerable resistance. Their color is usually yellowish-white.

Cavernous Angiomata.—These tumors are of but slight clinical significance, for when they are small, they do not give rise to distinctive symptoms. Only when they are large do they cause pressure symptoms, the latter condition being quite rare. Their usual size varies from that of a pinhead to that of a walnut. They occur in the aged, and are found in men oftener than in women.

Lymphangiomata.—These tumors are very rare, but have been found in the transverse fissure of the liver.

Adenomata.—These tumors are not infrequently met with in the liver, especially when the organ reveals cirrhotic changes. They are usually sharply circumscribed. Fibrous connective-tissue septa may be pronounced in the tumors, so that they are divided into a number of segments. The cells may be arranged in an acinous or tubular manner. Adenomatous tumors of the liver are classified by some authorities as belonging to the malignant growths. Hoppe-Seyler [1] classifies these tumors under the heading of malignant growths.

[1] Nothnagel's "Specielle Pathologie und Therapie."

MALIGNANT TUMORS OF THE LIVER.

Carcinoma of the Liver.—Cancer of the liver is a rare disease. Lichtenstern found that out of 10,007 cases of cancer, only 6% proved to be cases of carcinoma of the liver. It occurs most frequently between the ages of forty and sixty. This tumor is more frequently secondary, primary cancer being extremely rare. Of 258 cases of carcinoma of the liver collected by Hansemann [1] in the Berlin Pathological Institute, 25 were primary cancer of the gall-bladder, 6 were primary cancer of the liver proper (2 of these being questioned), and 2 were primary cancers of the large bile-ducts. Primary cancer of the liver is more frequent in the male sex, and it seems that cirrhosis, malaria, and the abuse of alcohol are predisposing factors. Cancer is more frequent in women than in men, and is most often secondary to such conditions as carcinoma of the breast, of the uterus, and the ovary. It may also result from carcinoma of other abdominal organs, particularly the stomach and pancreas. It has been claimed that traumatism, parasites, and infectious processes in general are predisposing agents. Carcinoma of the gall-bladder frequently follows chronic irritation from gall-stones. Siegert [2] found gall-stones associated with primary carcinoma of the gall-bladder in 95% of the cases, while in secondary cancer he found gall-stone in only 15% to 16% of the cases. Primary cancer of the gall-bladder is more frequently met with in females than in males, and it is probably explained by the frequent occurrence of gall-stone in the female sex. Gall-stones and obstructive jaundice also seem to predispose to carcinoma of the biliary passages.

Primary Carcinoma of the Liver.—The primary cancers of the liver may be quite large and massive, the liver being enlarged, and, as a rule, no cirrhosis accompanies the massive cancer. The capsule may be thickened, but is not adherent to the peritoneum. The primary cancer may be nodular, the cancer mass varying in size from a pea to a walnut, and in the majority of cases resembling the common variety of secondary carcinoma of the liver. The condition is also frequently accompanied by cirrhotic changes, with a decrease in the size of the organ. A third variety is known as the infiltration

[1] "Berliner klin. Wochenschr.," 1890, No 16.
[2] "Virchow's Archiv," 1893, Bd. CXXXII, p. 353.

primary cancer, in which a number of small cancerous masses are thickly infiltrated throughout the liver substance, the organ showing thickening of the capsule and adhesions of the peritoneum. Histologically, characteristics of carcinoma are present.

Secondary Carcinoma of the Liver.—This form of cancer is usually easily recognized macroscopically on account of the tremendous enlargement of the organ, with the appearance of carcinomatous nodules, which are noted projecting above the surface in many places. They are of a grayish or yellowish-gray color, and the upper surface of the nodule is irregularly umbilicated, and the mass is quite distinctly circumscribed. These carcinomata show a great tendency to degenerate, and it is on account of this that the central portions recede and produce the umbilicated appearance. The organ has been known to weigh eight kilograms. The infiltration may be so extensive that the parenchyma is scarcely visible. Upon section, the liver is found to be somewhat more resistant to the knife than normally on account of the infiltration of the new growth. Bile-stained and hyperemic areas may be noted on the cut surface. The carcinomatous masses are of a grayish or yellowish color upon section. The degeneration sometimes leads to cyst formation. Histologically, the characteristics of secondary cancer are noted. On account of the pressure of the new growth, there is atrophy of the liver-cells. It will also be noted that cirrhotic changes are common in secondary cancers.

Sarcomata of the Liver.—These are less frequently met with than carcinomata, and are usually secondary. Round-cell, spindle-cell, melanotic, and lymphosarcoma have been found as primary growths in this organ.

Symptoms of Malignant Tumors.—The general cachexia of malignant disease is present, which may, indeed, be the first symptom of the condition. With this there is the presence of a tumor affecting the liver or the hepatic area. Commonly hard nodules may be felt upon the surface of the liver. Symptoms of compression occur. There is pressure upon the portal vein or the gall-ducts, and frequently there is formation of secondary growths in the peritoneum, lungs, etc. Commonly, also, the symptoms of cirrhosis are present. The symptoms may be latent if the growth occur in the internal part of the liver; this is especially true of the adenomata which have been recognized only postmortem, the condition

not being suspected *intra vitam*. The general symptoms are those of disturbance of nutrition, anorexia, disgust for food, especially meats and fats, great emaciation, and the malignant cachexia. The subjective symptoms of the patient are compression and weight in the right hypochondrium, soon giving place to dull pain, which may radiate to the right shoulder-blade. If pressure be exerted by the growth upon the biliary region, jaundice is a symptom. If the pressure occur upon the portal vein or its radicals, ascites develops.

The blood reveals secondary anemia, this often being extreme. The erythrocytes are greatly reduced, and the hemoglobin markedly diminished; malignant leukocytosis occurs. Poikilocytosis develops if the anemia be marked. As a result of changes in the blood, hemic murmurs may be heard in the cardiac area, and edema is likely to occur.

Fever is commonly present; it may be either remittent or intermittent, and it may occur without the presence of complications. As a rule, the temperature does not rise above 102° F. When the malignant tumor is situated so as to occlude the common duct, symptoms of obstructive jaundice arise, with enlargement of the gall-bladder. When the cancer involves the gall-bladder primarily, this organ becomes enlarged, and is painful upon palpation; the cachexia and anemia develop, jaundice not necessarily being a symptom unless secondary nodules affect the larger bile-ducts, either in the liver or in the course of the common duct. The spleen shows no constant changes; as a rule, it retains its normal size; however, if the growth of the malignant tumor be slow, the spleen may show enlargement. The urine is usually diminished in amount, showing the presence of urobilin. Albumin and casts are occasionally found. In cases of melanosarcoma, melanin is found in the urine. Nervous symptoms occur, particularly late in the course of the affection. Pains in the region of the liver have already been referred to. Occasionally there are colicky pains. The patient is irritable and sleepless, and toward the close of the affection delirium and coma may occur, which are most probably due to cholemia.

Prognosis.—The prognosis is absolutely unfavorable. The duration of the disease is variable, commonly about one year. The course is rapid in the forms complicated by cirrhosis, and in adenomata the duration of the disease is much longer.

Treatment.—The treatment is expectant symptomatic.

DISEASES OF THE BILIARY PASSAGES.

OBSTRUCTIVE JAUNDICE.

Obstructive jaundice may arise from the following conditions: Catarrhal inflammation of the biliary ducts, stricture of the ducts, foreign bodies within the ducts, such as various parasites and stones; from tumors occupying the lumen, catarrh of the duodenum, and from pressure from without, due to tumors of the pancreas, the stomach, the liver, the omentum, or the kidney; and from aneurysms, large cysts, and enlarged glands. (See p. 31.)

TOXEMIC JAUNDICE.

This form of jaundice is called by some writers the hematogenous or the hemohepatogenous variety. It results from the introduction of any one of a number of poisonous substances into the stomach, and may arise in any of the infectious diseases. It sometimes occurs from snake-bites. (See p. 31.)

CATARRHAL JAUNDICE.

Etiology.—This often follows a gastro-intestinal catarrh due to indigestible food. It arises after a chronic catarrhal process, from chronic alcoholism. It sometimes follows cold and exposure in which the body has become chilled. Occasionally it may arise without assignable cause. Catarrh of the bile-ducts occurs in the course of the infectious diseases, such as malaria, typhus, and cholera. Poisoning by phosphorus also gives rise to catarrh of the bile passages. Gallstones, by irritating the mucous membrane, may give rise to catarrhal jaundice. It is more common in the early periods of life than in middle age, and more common in the male than in the female. In children the disease arises most often between the second and seventh years; previous to this age it is rare.

Pathology.—The mucous membrane lining the biliary passages in acute catarrhal jaundice can rarely be studied, as the disease never terminates fatally; however, it is easy to understand that should the epithelial cells lining these ducts

become swollen, granular, and be shed off and obstruct the onflowing bile, jaundice would follow.

Symptoms.—The symptoms are those of gastro-intestinal catarrh—a sensation of weight in the epigastrium, anorexia, coated tongue, nausea, vomiting, headache, vertigo, and occasionally slight fever. The bowels are constipated. Rarely does diarrhea arise. The urine is diminished in amount, and dark in color, containing sediment. After these symptoms have continued for several days or a week, jaundice develops, showing itself particularly in the skin and in the urine. The feces become light, sometimes clay-colored, owing to the absence of bile. These symptoms may last several weeks. When the tongue clears, the appetite returns and the signs of jaundice disappear, first in the urine and later in the skin. The duration in uncomplicated cases is from three to four weeks, but complete recovery may be protracted for several weeks. In from one-third to one-half of the cases enlargement of the liver takes place. As a rule, there is no pain in the region of the liver. Fever is usually present for a few days. When jaundice develops, the temperature becomes normal or even subnormal. With the development of marked jaundice the pulse becomes slow.

Prognosis.—As a rule, the prognosis is favorable. Only in the aged will long-continued jaundice produce dangerous symptoms.

Treatment.—The diet is important; it should consist of thin soups, of food that is free from fat, and of water in large amounts. Attention should be directed to the gastro-intestinal catarrh. Constipation should be corrected; for this purpose small doses of calomel followed by salines are useful. The patient should remain in bed. When jaundice appears, the alkaline mineral waters are of use, such as Vichy or Carlsbad; or phosphate of sodium may be administered in hot water.

ICTERUS NEONATORUM.

By this is meant a jaundice occurring in the new-born, in which speedy recovery takes place. It must not be confounded with jaundice occurring in the new-born due to septicemia or syphilis. Jaundice occurs in the new-born, according to statistics, in about two-thirds of the cases. It is much commoner in boys than in girls, and is likely to occur in children born after chloroform narcosis of the mother.

As a rule, the jaundice shows itself upon the second or third day, first upon the face, then upon the breast. The conjunctivæ become affected later than in adults. The jaundice may only last a few days—as a rule, to about the middle of the second week, but may last until the third or fourth week. Recurrences are rare. The general functions of the child are rarely interfered with. The urine is not discolored by bile, and, as a rule, albumin is not present. The bile pigments are absent. The feces are yellowish in color. The pulse is not slowed. Excretion of urine is increased. The prognosis in jaundiced children is as good as in those without jaundice. The jaundice does not require special treatment.

SUPPURATIVE CHOLANGITIS.

Suppurative infection of the biliary passages is rarer than simple catarrhal infection. It occurs particularly in old age as a result of gall-stones or parasites. It may occur as a complication or sequel of enteric fever, pyemia, or dysentery.

Etiology.—In the majority of cases cholangitis is due to microbic infection. Micro-organisms find their way into the biliary passages from the bowel. The entrance of micro-organisms into the biliary passages does not necessarily lead to suppurative inflammation, but very frequently results in a simple catarrhal process. The most common micro-organism producing cholangitis is the bacillus coli communis, either alone or combined with the staphylococcus albus or aureus, or with the streptococci. Parasites, such as ascarides, which find their way from the bowel into the biliary passages, may give rise to the affection. It also results from cancers of the duct.

Pathology.—The pathology is that of acute suppuration of the mucous membrane, which leads to obstruction of the flow of bile.

Symptoms.—These are rarely characteristic. Jaundice is not so intense as other symptoms, and may have preceded the suppurative cholangitis—as, for instance, from an attack of hepatic colic. Fever and enlargement of the spleen are important symptoms, the fever being of the remittent type, with evening exacerbations. Occasionally it may be intermittent. As in all septic conditions, the fever may be accompanied by rigors and sweating, and is often due to the occlusion of the common bile-duct by gall-stone. It has received the name of *Charcot's fever*, or *hepatic fever*. In some cases, and especially

if the gall-bladder be affected, pain in the hepatic area is a symptom. Commonly there are digestive disturbances, diarrhea, and vomiting.

Complications are pylephlebitis, septicemia, endocarditis, purulent meningitis, and, from extension from the gall-bladder, peritonitis.

The course of the disease is chronic. The onset is mostly insidious. If due to cholelithiasis, the symptoms manifest themselves after the signs of the passage of the biliary calculus have disappeared.

Prognosis.—The prognosis is unfavorable.

Treatment.—This consists in the thorough treatment of the cholelithiasis which is so frequently a cause of the affection. The remedies which are of most value are salicylic acid, salol, turpentine, and benzonaphthol. If the condition be due to ascarides, calomel and santonin are indicated. Surgical interference has proved effective, by means of opening and drainage of the gall-bladder.

CHOLELITHIASIS; GALL-STONES.

Gall-stones are composed of cholesterin and bile pigments, particularly bilirubin, with salts of calcium. Phosphorus, magnesia, and other elements are occasionally intermingled. Some mucus is usually present. An analysis of gall-stones shows them to consist of about 90% of cholesterin. The color of the stone depends upon the amount of biliary pigment present. If the stones consist almost entirely of cholesterin, they are nearly colorless or white; if a small amount of biliary pigment is present, they are of a golden-yellow hue; and if they possess much biliary pigment, they are of a golden-brown color or darker.

The consistency depends upon the constituents; thus, a fresh cholesterin stone may be easily broken up by the finger-nail. Freshly formed stones are usually soft. The shape depends upon the number of stones present and upon the position in which they are formed. If soft large stones are present, they show a flattened, smooth, mammillated and faceted surface. They may be rounded or elongated, with pointed ends, angular, or egg-shaped. When large, they are usually single. When they are small, several hundred may be present. As many as 7802 gall-stones were found in a case reported

by Otto. It has been estimated that gall-stones have been found in from 5% to 10% of all postmortem examinations.

They may occur at any age, and have been found in the newborn; they are, however, rarely found under the ages of twenty-five or thirty. They occur more frequently in women than in men, and it has been supposed that pregnancy is a predisposing cause, as is also the wearing of tight corsets, which may press upon the front of the liver, depressing the fundus of the gall-bladder. This, combined with lack of exercise, would account for the greater frequency in the female sex. Catarrh of the bile-ducts and gall-bladder may lead to stagnation of bile and to an increase in the amount of cholesterin. Farinaceous food may give rise to the formation of gall-stones, and in diseases in which nitrogenous food is largely partaken of, as in diabetes, gall-stones are rarely found. Gall-stones are formed most frequently in the gall-bladder. They may form in the larger gall-ducts, and rarely even in the smaller biliary passages of the liver.

Pathology.—According to Naunyn, the production of gall-stones takes place as follows: Lime salts and cholesterin originate from the mucous membrane of the biliary ducts. This is especially marked when catarrhal inflammation exists. Epithelial cells and various micro-organisms seem to form the nucleus for the formation of the stone. Micro-organisms have been demonstrated in gall-stone. Commonly a great number of stones are met with, but a single stone may be present either in the gall-bladder or in the common duct. They may be smooth, oval, mulberry-shaped, or polygonal, containing a number of facets. They vary greatly in size. A single stone may fill up the entire gall-bladder, and sometimes when many stones are present, they are as small as grains of sand. The stones are usually composed of large masses of cholesterin, which may be arranged in concentric layers, the outer portion of the stone being harder than the center. They also contain fatty and biliary acids, salts of lime and magnesium, copper, and iron. If obstructive jaundice continue for some time, cirrhotic changes usually develop in the liver. (See Biliary Cirrhosis.) Occasionally the stone may become firmly lodged in the common duct, and interstitial change take place in the walls of the duct so as firmly to encapsulate the stone. This may also occur in the gall-bladder. The stone may occupy the distal end of the common duct, or be in the ampulla of Vater, so as to form a ball valve

—that is, the quantity of bile distends the duct by the obstruction until the dilatation be so marked as to allow the escape of bile around the stone, and in some instances pushing it back, in this manner giving rise to intermittent jaundice.

Symptoms.—If the gall-stone remains quiescent and does not attempt to pass, symptoms do not arise. Occasionally there may be uneasiness in the right hypochondriac region, especially marked in changing the position of the body, or several hours after a meal. Rarely dull pains may occur in the right side of the epigastrium, which may also be present in the right shoulder-blade. The appetite may be variable. Occasionally nervous symptoms occur. The patient is irritable. Headache may be present, even neuralgias and migraine. If the abdomen be examined, the gall-bladder may be found enlarged. In rare instances stones may even be felt in the gall-bladder and be palpable through the abdominal wall. When the stone attempts to pass, distinctive symptoms are set up; the condition is then known as *hepatic colic*. The attack may follow a hearty meal or violent exercise, and usually begins with decided pain, which sets in suddenly (frequently about midnight or sometimes in the late hours of the afternoon). The pain is described as boring, stabbing, and is so severe that the patient usually cries out. The pain is generally situated in the right hypochondrium, in the epigastrium, or in some cases at a point corresponding to the region occupied by the gall-bladder. From here it may radiate upward into the chest or into the extremities, but most frequently through to the back and then into the right shoulder. Respiration, particularly inspiration, is painful; the breathing is therefore accelerated, superficial, and costal. The pulse is feeble, often slow, but may be rapid. Commonly, the patient flexes the right leg upon the abdomen to relax the belly wall. The pain is intermittent in character, and when the stone has passed into the intestine, it ceases abruptly. Accompanying the pain there is usually vomiting, at first of food, and later of biliary material. There are chilly sensations, followed by a rise in the temperature to about 103° F. or 104° F.; this is common. Sweating is rare. As a rule, the fever lasts but a few hours. One of the most important symptoms of gall-stones is the development of jaundice, which, as a rule, does not occur at once, but twenty-four hours after the beginning of the biliary colic. The urine is high-colored, containing bile elements; the stools are commonly clay-colored. The presence of gall-stones in the feces

is of great importance. Occasionally after well-developed symptoms of biliary colic no stone will be found in the feces upon careful examination. This may be due to several causes. Charcot has pointed out that during an attack of biliary colic the stone may fall back into the gall-bladder; this condition is rare. The stone may not pass completely, and remain in the common duct, or it may be broken up in the bowel. That this last-mentioned fact occurs Naunyn has proved.

The after-effects of biliary colic may arise from complications, weakness, loss of appetite, nervous symptoms, loss of sleep, etc. Usually, however, the patient is comparatively well after the attack. Death during an attack of biliary colic is very rare. Occasionally rupture of the duct has taken place, bile being poured into the peritoneal cavity. If the stone does not pass, and remains in the common duct, cholangitis occurs, which may give rise to characteristic symptoms known as Charcot's intermittent fever or hepatic fever (described under Suppurative Cholangitis; see p. 540).

The following **complications** may occur: Intestinal obstruction; hemorrhages; localized peritonitis; ulceration of the bile-ducts, establishing a fistula between the common duct and the intestines; stricture of the cystic or common bile-duct; abscess of the liver; empyema of the gall-bladder; suppurative cholangitis; extravasation of bile into the peritoneal cavity; cancer of the gall-gladder, and others.

Diagnosis.—Differential diagnosis must be made between biliary colic and appendicitis, hysteria, renal colic, and lead colic. The diagnosis of biliary colic depends upon the paroxysmal sharp pain in the right hypochondrium, radiating to the back and the right shoulder-blade; and upon the presence of vomiting and collapse, followed by jaundice. If the stone be arrested in the common duct, intense persistent jaundice and rarely the symptoms of Charcot's fever may be noted. Appendicitis may be differentiated from biliary colic by the fact that the pain is not so paroxysmal, but is more continuous, and is often limited to "McBurney's point." There is usually the presence of tumor in the right iliac fossa; pain rarely radiating to the right scapula. Jaundice, as a rule, does not occur. In hysteria the general neurotic temperament of the patient must be taken into account; the globus hystericus, the absence of collapse, and jaundice are important points. Renal colic is more closely associated with

urinary symptoms, the pain radiating into the testicle and along the right genitocrural nerve. In lead colic the pain is persistent. It is not limited to the region of the gall-bladder, and does not radiate, and, as a rule, there is a blue line upon the gums; paralysis of the extensors may be present.

Prognosis.—Generally the prognosis of the individual attack is favorable. The prognosis is less favorable when hepatic fever occurs or when there are signs of a circumscribed peritonitis or empyema of the gall-bladder. If sudden rupture occur, with the effusion of bile into the peritoneum, the prognosis is hopeless. If complicated by carcinoma, the prognosis is bad.

Treatment.—Prophylaxis.—This consists of appropriate diet, exercise, and general favorable hygiene. In women, tight lacing should be avoided. Warm baths, regular exercise in the fresh air, etc., should be recommended. The patient should avoid indulgence in sweets and starchy foods. Constipation should be avoided; this may best be done by the use of waters, such as Carlsbad. Massage has been strongly advised.

Treatment of the Attack.—Hot fomentations should be applied over the upper part of the liver. If the pain be severe, whiffs of chloroform may be inhaled; however, in the majority of cases, relief only follows the hypodermic use of morphin. If the condition is not relieved by medical means, and the jaundice remains persistent, especially if symptoms of Charcot's fever occur, surgical interference should be resorted to. The indications for operation are as follows: In recurring hepatic colic without jaundice, with or without enlargement of the gall-bladder, especially if accompanied by great pain; in jaundice following pain and symptoms of Charcot's fever; in empyema of the gall-bladder; in peritonitis starting from the right hypochondrium; in cases in which adhesions remain which may prove painful; in cases of fistula.

DISEASES OF THE HEPATIC VESSELS.

DISEASES OF THE PORTAL VEIN.

Obstruction of the portal vein may result from the growth of tumors pressing upon this vessel. The most common causes of compression are tumors of the stomach, pancreas, and the mesentery, and enlargement of the retroperitoneal glands, or from the liver itself. It may result from thrombosis, this being due to roughening of the endothelial coat, and is often of syphilitic origin. Obstruction may result from cirrhosis, but complete obstruction from this cause is rare. Obstruction has been known to result from great numbers of the distoma hematobium in the finer capillaries. The thrombus may organize, and the portal vein has been known to be completely occluded by fibrous connective tissue, nothing but the fibrous cord remaining, this condition being called *pylephlebitis adhæsiva.*

Symptoms.—Narrowing of the portal vein through thrombosis may give rise to symptoms which show the sudden onset occurring in the course of cirrhosis of the liver, chronic peritonitis, or tumors of the abdomen. They consist in the sudden appearance of symptoms due to stasis, marked epigastric pain, with vomiting and diarrhea, hematemesis and enterorrhagia. In the course of a few days ascites and enlargement of the spleen occur. The ascites soon leads to edema of the lower extremities, as a result of the pressure upon the vena cava. A caput medusæ forms, and even cutaneous edema may occur. If, after paracentesis abdominis—which soon becomes necessary—has been performed, the liver is examined, it will be noted that it has decreased in size. Occasionally, jaundice occurs. In rare instances these symptoms just enumerated may come on gradually, and may closely resemble the development of atrophic cirrhosis. The urine is decreased in amount, and, according to some authorities, glycosuria occurs. This is explained by the fact that the sugar-forming elements are carried through the collateral circulation to the heart, and then through the general circulation, reaching the kidneys without having undergone metabolistic change in the liver.

Duration.—The duration of the disease varies from a few

days to several years. If profuse hemorrhages occur, the disease may rapidly prove fatal.

Prognosis.—The prognosis is always unfavorable, the only cases amenable to cure being those due to syphilis with gummata formation.

Treatment.—The cases in which syphilis is suspected should be treated by mercury and iodid of potassium. From other causes the treatment is purely symptomatic.

INFLAMMATION OF THE PORTAL VEIN.—PYLEPHLEBITIS.

ACUTE PYLEPHLEBITIS.

Synonyms.—Suppurative pylephlebitis; ulcerative pylephlebitis.

Etiology.—In rare instances an acute inflammation of the portal vein may be due to foreign bodies, which have traversed the wall of the bowel, and in this way reached the portal vein. It may also result from a purulent exudate from the lymph-glands reaching the portal vein. More frequently the inflammation begins in the terminal branches of the portal vein, which are in close relation to the abdominal viscera, particularly those of the bowel, especially the appendix vermiformis. Fistula in ano, hemorrhoids, and carcinoma may cause the condition. It may result from trauma, such as might arise from the careless introduction of the rectal tube, and may follow abdominal operations. It may also result from inflammation of the uterus and its appendages or of the bladder, ulceration of the stomach, splenic abscesses, mesenteric abscesses, purulent pancreatitis, and, in the new-born, from inflammation around the umbilicus. It may result from empyema of the gall-bladder, and from other inflammatory conditions of the biliary passages.

Pathology.—At first the veins become thickened and the walls infiltrated with an inflammatory exudate; a thrombus may then result; often it is broken up, producing emboli, which lodge in the liver, giving rise to small abscesses. The walls of the vein may reveal ulcerated areas, and in some instances may rupture. This acute inflammatory infection is always of bacteriologic origin. Large solitary abscesses, as well as small ones, may arise in the liver from this cause.

Symptoms.—The symptoms are those of septic processes

joined to those of the primary affection. There is an irregular, high temperature, either intermittent or remittent in type, accompanied by chills, sweating, and collapse. Occasionally, the liver is enlarged, owing to cloudy swelling. The spleen is enlarged in the majority of cases. There is anorexia, vomiting, diarrhea, jaundice in some cases, and, toward the close of the affection, enterorrhagia. The urine is diminished in amount and is albuminous.

Duration.—The duration of the disease is from two to six weeks.

Prognosis.—The prognosis is always unfavorable.

Treatment.—The treatment is expectant symptomatic.

CHRONIC PYLEPHLEBITIS.

As in other blood-vessels, sclerotic changes may take place in the portal vein, with thickening of the intima or with calcareous infiltration. The vessel wall becomes inelastic, the lumen narrowed, and, as previously stated, may become occluded (pylephlebitis adhæsiva). Syphilis is a cause of this condition. Weigert describes tuberculosis of the portal vein.

Disease of the Hepatic Artery.—The hepatic artery may be the seat of aneurysm or of sclerotic changes.

Disease of the Hepatic Veins.—Stenosis may result from compression through new growths or cicatrices, most often of syphilitic origin, which develop in the liver structure, or from inflammation of the hepatic vein, which is rarely primary. Thrombosis may also occur and give rise to occlusion. Emboli may lodge in the hepatic veins as a result of a regurgitating blood stream from the right heart, particularly the right auricle, sometimes during forced expiratory movements, as coughing.

Inflammation of the Hepatic Veins.—This may be either acute or chronic. Acute inflammation is most frequent, and is due to suppurative inflammation of the liver, such as suppurative pylephlebitis, to echinococcus cysts, or to purulent cholangitis. The chronic variety is most often due to syphilis. There are no symptoms by which this condition can be recognized.

DISEASES OF THE PANCREAS.

INFLAMMATION OF THE PANCREAS.

ACUTE HEMORRHAGIC PANCREATITIS.

The whole or only a part of the pancreas may be involved by this process, in which the inflammation is combined with hemorrhage.

Etiology.—Trauma may be a cause. Chronic alcoholism has been noted in a number of the cases; it is, however, most commonly due to an extension of inflammation from the duodenum to the pancreas through Virsung's duct.

Pathology.—The organ may be enlarged and deeply stained with blood. On section, large areas or only punctiform ones may be seen. Extensive fatty necrosis of the pancreas and surrounding structures, as well as inflammatory changes, are noted.

Symptoms.—As a rule, the symptoms appear suddenly. Severe pains in the epigastrium, in the region of the umbilicus, nausea, vomiting, with constipation and signs of rapid collapse, are pronounced symptoms. There is great uneasiness, marked rapidity of the pulse (tachycardia),—140 to 160 per minute,—dyspnea, subnormal temperature, and rapid loss of strength; death occurs from exhaustion in from a few hours to a few days.

PURULENT PANCREATITIS (ABSCESS OF THE PANCREAS).

(a) **Primary Pancreatitis.**—As etiologic factors, alcoholism, pregnancy, suppression of the menses, and poisoning from mercury have been given. Trauma may also be a cause. Necessarily, pyogenic organisms must find their way into the pancreas. The disease occurs much more frequently in men than in women, and arises most commonly between the ages of twenty and thirty.

Pathology.—The organ is enlarged, and abscesses are found scattered throughout, or they may be more numerous in certain parts. The necrotic process may destroy a part or the whole organ, and through rupture the pus may find its way into the peritoneal cavity.

Symptoms.—As a rule, the symptoms occur suddenly in the course of or following hepatic colic or digestive disturb-

ance. There is violent pain, coming on suddenly in the epigastrium, which is localized and does not radiate from the abdomen. However, this is not invariable, as cases have been recorded without pain. In rare instances pains may show themselves in the splenic region, the spleen being tender, pain being elicited in this area upon palpation. Nausea and vomiting, with eructations, are almost constant symptoms, the vomited material often being bile-stained. With these symptoms there is rapidly oncoming and marked prostration. Fever of an irregular type, accompanied by rigors, is present, but in rare instances the fever may be absent. As a rule, constipation occurs. In rare instances diarrhea is present, which later may become profuse. Occasionally, diarrhea may alternate with constipation. The feces may contain blood and fetid pus, the abscess having ruptured into the bowel. Albumin and fat may be present in the stool. The liver is enlarged, and also the spleen, but to a less extent. There is considerable tympany of the abdomen. In some cases fluid may be found in the belly, or there may be other signs of accompanying peritonitis. Upon physical examination the epigastrium is resistant, or even a tumor may be found in the abdomen. The urine is usually of low specific gravity—1002 to 1005. Peptone, albumin, sugar, and indican have been found present. Jaundice occurs in about 25% of the cases. In cases that run a chronic course marked emaciation and petechia, or other purpuric manifestations, may occur.

Treatment.—The treatment is surgical.

(b) Secondary Acute Purulent Pancreatitis.—The secondary variety may arise from inflammation of surrounding organs from continuity of structure, also in the course of pyemia, puerperal fever, and from malignant disease of the pancreas or other surrounding organs. Rarely it may result from acute peritonitis.

The symptoms are those of the underlying affection, linked with those just enumerated in the primary variety.

GANGRENOUS PANCREATITIS.

Gangrenous pancreatitis may result from suppurative inflammation or in the course of chronic pancreatitis. Necrosis may occur from hemorrhage. It is doubtful whether fat necrosis of the pancreas ever leads to gangrene; some authorities, however, have recorded such instances. Gall-stones may

give rise to the affection, and it may be produced from unknown causes.

Pathology.—The pancreas may be partially or entirely involved. Sometimes the organ is of a brownish-red or green color, emitting a stench which is common to gangrenous affections. Rupture into the intestine with discharge of the gangrenous mass has been recorded. General peritonitis does not usually occur; more commonly a localized process is encountered.

Symptoms.—The causes which give rise to gangrene of the pancreas are so varied that the symptomatology necessarily depends upon the etiologic factors. As a rule, violent pains, which are limited to the epigastrium and radiate in all directions, are common. Nausea with vomiting, tympanites, tenderness, symptoms of collapse, such as a rapid pulse, irregular fever, with a dry tongue, etc., are present. Obstruction of the bowel is a common symptom. If rupture occurs in the retroperitoneal space, a tumor may be noted upon palpation.

Diagnosis.—The diagnosis must be made by exclusion.

CHRONIC INDURATIVE PANCREATITIS.

This may arise from two groups of causes—from indurative inflammatory processes, as a result of disease of the vessels, such as arteriosclerosis and endarteritis obliterans, especially as a result of syphilis and alcoholism; and, secondly, from chronic indurative inflammatory processes, or obstruction and narrowing of the pancreatic duct. The organ may be small and very hard. Rarely is it larger than normal. Sclerosis and atrophy of the pancreas are of interest, as they bear an etiologic relationship to diabetes mellitus; and, also, if the sclerotic process be present in the head of the organ, obstruction of the common bile-duct from contracture may result.

TUMORS OF THE PANCREAS.

BENIGN TUMORS.

These tumors are extremely rare in this organ. Adenomata have been met with and recorded in recent medical literature. Extirpation of these tumors has resulted in recovery.

MALIGNANT TUMORS OF THE PANCREAS.

Carcinomata of the Pancreas.—Carcinomata of the pancreas are rare. In 23,611 autopsies collected by Biach,[1] of diseases of the pancreas, 29 carcinomata of the pancreas were found. Primary carcinoma is most frequent in the head of the pancreas, next in frequency being the body, and the rarest situation being in the tail. Secondary cancers of this organ are also encountered. The scirrhous form is most common, the encephaloid variety being rare. Males are more frequently affected than females, the most common age being between the fortieth and the seventieth years.

Cachexia, as in malignant disease of other organs, is also noted here, with progressive increasing weakness and loss of weight; the presence of a tumor in the region of the pancreas, with pain, which may be paroxysmal, are symptoms. Fatty stools may occur. If the cancer affect the head of the pancreas, jaundice is likely to appear. Diabetes is met with in cancer of the pancreas. On examination of the patient, the tumor, which is tender upon pressure, may be felt. The disease invariably terminates fatally, usually running a rapid course.

Sarcoma.—Primary sarcoma of the pancreas is an exceedingly rare affection, but secondary melanotic sarcoma is more common.

Treatment of Malignant Tumors of the Pancreas.—The treatment consists in relieving the pain by the administration of opium in some form. Operation should be advised if the condition be diagnosticated early.

PANCREATIC CYSTS.

They may be due to blocking up of the pancreatic duct, giving rise to retention cysts. Proliferation cysts and retention cysts of the smaller ducts are also encountered. Inflammatory changes of the pancreas may give rise to cysts. The cyst may develop so that the stomach is pushed upward, or it may form above the lesser curvature, displacing the stomach downward; or it may be so large as to lie beneath the colon; and occasionally it is found in the left hypochondrium.

Symptoms.—Frequently the condition can not be diagnosticated. As a rule, a tumor is found occupying a position between the liver and spleen. There is rapid emaciation, and

[1] Nothnagel's "Specielle Pathologie und Therapie."

paroxysmal attacks of pain may occur. Symptoms of diabetes, such as glycosuria, are likely to occur. An exploratory puncture may be undertaken in doubtful cases, when, if fluid from a pancreatic cyst be found, it will contain epithelial cells, red blood-cells, and pancreatic ferments. It is strongly albuminous, in contrast to the fluid from echinococcus cysts, which does not contain albumin.

Treatment.—The treatment is surgical.

PANCREATIC CALCULI.

Pancreatic calculi are rare. There are usually many stones, and they scarcely ever attain a large size. The largest stone recorded was found by Schupmann; it was 2½ inches long and from 5 to 6 lines in diameter. It weighed two ounces. The stones are usually of a grayish or yellowish-white color, and rarely darker. The origin of the stone has by no means been satisfactorily explained. The calculi are composed chiefly of carbonate and phosphate of lime, and contain cholesterin plates. They give rise to cystic formation of the ducts and to interstitial changes in the organ. The affection is most common in males, and arises most often between the ages of thirty-five and forty-five.

Symptoms.—If the stone be firmly situated, no symptoms arise, but upon passage colicky pains develop, accompanied by nausea, vomiting, and symptoms of collapse. Diarrhea, with the occurrence of fat in the stools, and glycosuria have been noted.

Diagnosis.—The diagnosis depends upon the appearance of the characteristic concretions in the feces, the diabetes, the fatty stools, the colicky pains, salivation, and jaundice.

Treatment.—It has been experimentally determined that pilocarpin increases the flow of the pancreatic juice; hence, hypodermics of pilocarpin are indicated in pancreatic colic. For the pain itself, opium is necessary; and hot fomentations to the epigastrium in the region of the pancreas are grateful to the patient. If abscess or cyst formation take place, surgical interference is necessary.

HEMORRHAGE INTO THE PANCREAS.

Etiology.—Hemorrhage may result from trauma. It is occasionally due to a dilated right heart, from the general venous stasis which is apt to follow this condition. It is

believed by some to be of nervous origin. The hemorrhage may be either large or small, depending upon the cause.

Symptoms.—Pain in the epigastrium, corresponding to the region of the pancreas, with the appearance of shock, frequent vomiting, and occasional diarrhea, are the diagnostic criteria. The pulse becomes rapid and feeble, and the temperature may be subnormal. Recovery may take place from small hemorrhages; profuse hemorrhages are commonly fatal.

Treatment.—The treatment consists in absolute rest, the use of opium in some form, and the local application of ice.

FAT NECROSIS OF THE PANCREAS.

This affection is associated with some of the diseased conditions of the pancreas. Yellowish necrotic areas varying from the size of a miliary tubercle to that of a pea, or sometimes as large as a hen's egg, are found in the pancreas, in the surrounding tissues, and sometimes in the abdominal wall. They are most often noted in the interlobular spaces of the pancreas. Other seats in which these fat areas can be found are in the omentum, the perirenal fat, the mesentery, the abdominal wall, and occasionally in the bone-marrow.

Experiments performed by Katz and Winkler[1] show that by ligation of the pancreatic duct, and also by ligation of the glands at various parts, fat necrosis was produced, accompanied by an enormous leukocytosis. In one case, in which a dog was operated upon, there were 12,500 leukocytes per cubic millimeter; two days following the operation there were 310,000 leukocytes. The dog died upon the ninth day after the operation; marked necrosis of the pancreas was found, but no purulent material.

The causes of fat necrosis are not definitely understood. It has been suggested by Langerhans that it is due to the liberation of the fat-splitting ferments of the organ into the digestive tissues. Bacteriologic examinations of the necrotic areas show that they are sometimes sterile and sometimes infected by various organisms. Diabetes mellitus has been found associated with fat necrosis.

Symptoms.—The symptomatology shows wide variations. The disease may exhibit the symptoms of acute intestinal obstruction, or the symptoms of retroperitoneal tumor, or of perforating peritonitis. In other cases the symptoms may

[1] Oser, "The Diseases of the Pancreas," vol. XVIII of Nothnagel's "Specielle Pathologie und Therapie."

resemble cholelithiasis, with severe colic and collapse, or the symptoms may resemble a toxemia due to a severe infection or active poison.

RUPTURE OF THE PANCREAS.

This is an exceedingly rare condition, as the pancreas is so deeply situated in the abdomen, being well protected against trauma; the force must be applied directly backward in the upper part of the abdomen. The symptoms are those of shock, similar to those in severe internal hemorrhages.

PART V.

DISEASES OF THE KIDNEYS.

ACTIVE CONGESTION.

Synonyms.—Active hyperemia; hyperemia.

Active hyperemia is the first stage of inflammatory conditions of the kidney; it occurs when one kidney is doing work for the other (compensatory hyperemia), also in hypertrophy of the left ventricle, from diabetes mellitus and diabetes insipidus, and in certain diseases of the nervous system, particularly of the medulla oblongata and the sympathetic nerves; lastly, from certain irritants and diuretics, such as cubebs, turpentine, etc. The organ is large, of a dark red color, and upon section blood drips from the cut surface; it is also slightly more friable.

PASSIVE CONGESTION.

Synonyms.—Passive hyperemia; congestion; cyanotic induration.

Etiology.—This condition is most commonly due to valvular heart disease when failing compensation develops, the blood being held back in the larger veins, so that it can not escape from the organ. It may also occur from mechanical compression of the venous trunks, such as a tumor pressing upon the renal veins or upon the vena cava.

Pathology.—The kidney is larger than normal, especially in its shortest diameter, it therefore being somewhat rounded. Its color is reddish-blue (cyanotic); the capsule is found to retract slightly when the knife is inserted into the organ, and is easily stripped. Upon section the knife meets with more resistance than normally, owing to the kidney being indurated.

The cut surface freely drips dark colored blood, and the Malpighian bodies and the pyramids stand out prominently, and are deeply stained, being in marked contrast to the surrounding kidney structure. Microscopically, it will be found that the veins are dilated, and frequently sclerotic changes are found, particularly about the Malpighian ducts. If the congestion be long continued, the kidney becomes smaller, owing to marked interstitial change. The color then becomes lighter, often not being so deeply stained as the normal organ. The surface is irregular; the fibrous capsule strips with difficulty; the organ is very tough (cyanotic induration), and cuts with much resistance. Upon microscopic examination it will be found that there is a marked increase in the fibrous connective tissue between the tubules, and the Malpighian bodies may be replaced by the interstitial tissue.

Symptoms.—The diagnosis of this condition must be made principally through the urine. The urine becomes diminished in amount, the color darker than normal; it is strongly acid in reaction, with a decided increase in the specific gravity—from 1025 to 1030. In consequence of the diminished amount of fluid secreted, the urates deposit in large amounts, showing the characteristic "brickdust" sediment. The urine may contain bile pigments, particularly urobilin. Sooner or later albumin shows itself, usually in small amounts. Tube-casts, particularly hyaline, occur with the albumin, and there are a few leukocytes. Exceedingly rarely, erythrocytes are present, and then only in small amounts. These probably are found in the urine as a result of the rupture of the blood-vessels into the tubules. These changes which occur in the urine are really the results of congestion due to lessened arterial and increased venous pressure. Fever does not occur with this condition. Accompanying this urinary affection there is, as a rule, marked dyspnea and cyanosis, gastro-intestinal catarrh, enlargement of the liver with jaundice, hemorrhoids, headache, and dropsy, these symptoms all being due to the same cause—ruptured compensation in valvular heart disease.

Prognosis.—The prognosis is usually favorable, except in those cases in which interstitial changes take place.

Treatment.—Efforts should be made to increase the muscular activity of the heart. Cardiac tonics are indicated, digitalis deserving the front rank. If digitalis be contraindicated, strophanthus, caffein, and spartein may be used as substitutes. The patient should remain in bed. Active purgation should

be instituted, this being best accomplished by calomel, which is at the same time a diuretic, and may be followed by salines. The diet should consist of milk.

ACUTE DIFFUSE NEPHRITIS.

Definition.—Acute diffuse nephritis is an acute inflammation of the kidney involving the entire anatomic structure of the organ, although in the majority of cases the epithelial cells are principally affected.

Synonyms.—Acute parenchymatous nephritis; acute tubular nephritis; acute Bright's disease; acute desquamative nephritis; acute exudative nephritis.

Etiology.—Acute nephritis is due to the direct irritation of some toxic substance brought to the kidney from the blood; thus, the infectious diseases frequently give rise to it,—*scarlet fever*, diphtheria, yellow fever, erysipelas, enteric fever, malaria, smallpox, croupous pneumonia, acute rheumatic fever, and in some of the more chronic infectious diseases, as syphilis and pulmonary tuberculosis. The disease may result from traumatism and suppuration. An important cause is exposure to cold and wet. The disease arises occasionally in the course of pregnancy. Finally, certain drugs give rise to nephritis. The disease has followed the internal use of cantharides, chlorate of potash, turpentine, squills, carbolic acid, corrosive sublimate, and some of the balsams. Alcohol exceedingly rarely gives rise to acute nephritis.

Pathology.—The changes in the kidney vary greatly, depending upon the intensity of the irritation and upon the vascular supply of the organ. The kidney is always enlarged, the size, however, varying somewhat. The fibrous capsule is tense, and when incised, it is found to retract slightly. It strips quite easily unless chronic interstitial changes preceded this condition, binding the capsule to the kidney substance. The surface of the kidney in the early stages is smooth and of a dark, reddish-gray color. Upon section the organ is found to be of a lessened consistency, and quite friable. The cut surface drips blood, the pyramids and the Malpighian bodies are found deeply stained, and the entire surface of a deeper color than normal in the early stages; later, this color changes to a yellowish-gray. It will also be noted that the entire width of the cut surface is increased, this

being most marked in the cortical portion, so that the cortex equals one-half of the width.

Anatomically, three varieties have been differentiated: The first one, which is very red, is called the hyperemic or hemorrhagic; the second is a pale form; and the third is a mottled, which should be classed as an intermediate condition between the pale and the hyperemic.

The *microscopic* examination shows that the epithelial cells lining the tubules are affected. When the epithelial cells of the kidney, both of the glomeruli and the remaining parts of the tubules, are affected, the condition should be properly spoken of as *parenchymatous nephritis.* If the changes be more pronounced in the glomeruli, it may be spoken of as *glomerulonephritis;* or if the changes be more marked in the tubules, *tubular nephritis;* and if the interstitial parts also show acute inflammatory lesions, as well as the tubules and the glomeruli, the condition is known as *diffuse nephritis.*

The epithelial cells lining the tubules at first become swollen and granular (cloudy swelling), so that the lumen of the tubule is decreased in diameter and the width of the tubule is increased, therefore giving rise to swelling of the organ. Later, the epithelial cells may become very irregular in outline, and show fatty changes, or sometimes hyaline degeneration. The nuclei of the cells share in the degenerative process, and in stained preparations they are with difficulty demonstrated, or the staining reaction becomes somewhat modified. In the lumina of the tubules hyaline and granular material, loose epithelial cells, leukocytes, and red blood-corpuscles may be noted. It will be found that the larger or terminal tubules rarely show changes in the epithelial lining, but the lumina are filled with casts, red blood-cells, leukocytes, and degenerated epithelial cells which have been washed out from the smaller tubules. The interstitial part of the organ will show that the blood-vessels are dilated, and leukocytic infiltration is always found. Hemorrhages into the kidney substance occur, particularly in acute *diffuse* nephritis.

Symptoms.—The disease may begin suddenly or gradually. A sudden onset is more likely to take place after exposure to cold or wet, or in the course of one of the infectious diseases, particularly scarlet fever, the patient having several rigors, followed by fever, and pain in the loins, which is increased by pressure, and general malaise. Edema develops rapidly; the urine becomes scanty and high-colored, and

uremic symptoms, such as vomiting and convulsions, occur. This is a rare form of onset. The mode in which the disease commonly shows itself is in the gradual onset of all the symptoms. The urine becomes scanty and high-colored. In fact, the symptoms may be so mild that attention is first directed to the case by the development of uremic phenomena. Ordinarily, after several days some slight facial edema, particularly about the eyelids, makes its appearance. As a rule, this is noticed only at the onset, in the morning upon awakening, and passes away in a few hours. Later in the course of the disease it becomes permanent, and the dropsy becomes more general. In many cases edema is the first symptom of the disease. As the affection progresses, the digestive organs begin to manifest symptoms. Nausea occurs, which soon passes into constant vomiting. Constipation, which has at first been present, gives place to diarrhea. There is dull headache, with pains in the back and loins, edema of the feet, dryness of the skin, and dyspnea. The face becomes pale and puffy, and is quite characteristic. The urine is markedly diminished in quantity; even anuria may occur. Fever, as a rule, is absent; when present, it is due to the primary condition causing the nephritis. When the disease is arrested, the edema gradually disappears, nausea and headache cease, the skin becomes moist, the urine is more profuse, and the patient gradually recovers. This is the rule in nine-tenths of the cases. If this does not take place, the albumin and the edema do not entirely disappear, the patient is troubled with dyspnea and weakness, and the acute form merges into the chronic variety. In cases that terminate fatally the symptoms increase in severity; nervous phenomena develop, such as convulsions, followed by coma; edema of the lungs, or pericarditis. The three terminations of acute nephritis are, then, complete recovery, merging into the chronic variety, and death. The exact diagnosis of acute nephritis can be made only by an examination of the urine.

The Urine.—The urine is always decreased in amount; it may be as little as 50 c.c. in twenty-four hours, but even such cases have resulted in recovery. The color is dark red and the urine is turbid, blood being present. The amount passed during the night generally shows less blood than that passed during the day. The specific gravity is from 1020 to 1030, and upon testing the urine large quantities of serum-albumin are found, from ½% to 1%. The quantity of urea is usually less

than normal. Under the microscope, hyaline, granular, and epithelial casts are noted, with renal epithelium, red blood-cells, and granular matter. Hematoidin crystals, either free or in cylinders, are noted, as are also various micro-organisms. Oxalate of lime and uric acid are not infrequent. In some cases blood-casts are found.

Dropsy.—Next to the urine in importance is dropsy. It shows itself first in the face beneath the eyelids, then about the ankles, about the tibia (pretibial edema) and other parts of the body. Serous effusions also take place about the loose tissues of the genitals and in the serous cavities. The facies of the patient is characteristic. The face is pale and puffy, the eyelids are swollen, and the skin is dry and coarse.

The Pulse.—As a rule, the pulse is not markedly accelerated; it may even in rare cases become slow—from 35 to 50; the sphygmograph, however, reveals high tension without hypertrophy of the left ventricle.

The Gastric Symptoms.—Nausea, vomiting, and diarrhea occur, although not so frequently as in the chronic forms. The nervous symptoms are most often due to uremia.

Diagnosis.—The direct diagnosis depends upon the history of the case, the general appearance of the patient, and the examination of the urine, in which blood, with albumin and casts, are present.

Prognosis.—The majority of cases recover entirely; some few, however, merge into the chronic variety. Death is a rare result. A small, frequent, soft pulse is an unfavorable sign, as is the development of uremia. Inflammatory complications and the presence of fluid in the serous cavities are unfavorable symptoms. In favorable cases recovery takes place within four weeks; however, many cases may go on for months and still recover. Cases, as a rule, which terminate in death are of short duration.

Treatment.—The patient should be put to bed, warmly clad, preferably with woolens next to the skin. The temperature of the room should be from 68° F. to 72° F., with good ventilation. Daily sponging with warm water and general friction of the skin are of use. Sweating should be induced by the use of the hot pack or by other means; this is recommended by many. The diet is most important. The best food for the acute and subacute cases is milk; three quarts during the day is sufficient. Some of the alkaline mineral waters, as Seltzer and Vichy, may be mixed with the milk. If

the milk does not agree, kumiss or buttermilk may be substituted. Water should be administered freely. Calomel in fractional doses is useful. This may be followed by a saline, even if there be no tendency to constipation. Dry cups are occasionally employed to relieve the kidneys, with the hope of exciting the flow of the urine. Liquor ammonii acetatis, citrate of potash, and benzoate of soda may be given as diuretics. Diuretin in 15-grain doses, administered three or four times during twenty-four hours, is of use in some cases. Digitalis, as a rule, is not indicated. In convalescence gentle exercise may be allowed, great precautions being taken against cold. Alcohol and tobacco should be avoided. For the anemia, which may persist, iron in some form is of value. The treatment of uremia is described on page 588.

CHRONIC DIFFUSE PARENCHYMATOUS NEPHRITIS.

Synonyms.—Diffuse parenchymatous nephritis; chronic desquamative nephritis; chronic tubular nephritis.

Etiology.—This is the most common form of renal disease, three varieties being found at autopsy, which differ from one another in their gross and microscopic appearances. In all, however, the change begins in the epithelial cells of the kidney, being a parenchymatous inflammation. The symptomatology of the three conditions being somewhat different, the clinician is often enabled to determine with which form he is dealing.

The varieties of chronic parenchymatous nephritis are: (1) The variety known as the *large white kidney;* (2) the *chronic hemorrhagic kidney;* and (3) the *mottled or cirrhotic kidney.*

The affection is most common between the twentieth and the fiftieth years of life, the disease rarely occurring in early adult life or in the aged, the male sex being more frequently affected than the female. Heredity is an important element in the causation of the disease. It is particularly a disease of the poorer class, especially those living amidst unsanitary surroundings, in damp buildings, etc., and of those who are exposed to the vicissitudes of the weather. Alcoholism is perhaps the most important etiologic factor, the disease occurring most frequently in the steady drinker who takes his daily quantum of alcohol, and not in the one who becomes periodi-

cally intoxicated. In general terms it may be said that when acute nephritis has lasted longer than eight months, the disease may be considered chronic. The chronic forms are likely to develop from the acute variety, especially that form due to pregnancy or to scarlatinal or malarial infection. The disease occasionally occurs in the course of chronic endocarditis. According to Bamberger, 7% of the cases of chronic nephritis may be attributed to endocarditis. The disease also occurs in the course of pulmonary tuberculosis, chronic suppurative processes, and syphilis, and in the gouty and the lithemic.

Pathology.—The various forms of chronic parenchymatous nephritis have this in common, that the kidney is usually increased in size. The extent of the involvement varies considerably, so that one form often merges into another—that is, a distinct line of demarcation can not well be drawn between the various varieties. In chronic parenchymatous nephritis the epithelial cells, especially those of the cortical portion of the kidney, are almost exclusively involved, the lesion varying from cloudy swelling to marked fatty degeneration. The epithelium lining the tubules becomes desquamated and irregular in outline, often blocking up the opening of the tubule, which may contain leukocytes, epithelial cells, etc. A certain amount of interstitial change is always present. In some areas there are found numerous cells infiltrated, there being some leukocytes, many round cells, and occasionally red blood-cells; edema is also noted in the interstitial process. The Malpighian bodies share in the pathologic change; in many cases the epithelial cells become granular, fatty, and desquamated. Between the blood-vessels making up the tuft there is an albuminous exudation which presses upon them. Bowman's capsule is often thickened. The interstitial change in chronic parenchymatous nephritis is never diffuse, but scattered through various portions of the substance.

Pathology of Large White Kidney.—This is the rarest of the various forms of chronic nephritis. Amyloid disease of the kidney resembles the large white kidney. An error may arise in differentiating between the gross appearance of these two conditions, which is somewhat similar; however, microscopic and chemic tests prove their individuality. This kidney is markedly enlarged; the fibrous capsule strips readily, leaving a smooth surface beneath. The substance of the kidney is found to be somewhat doughy, cutting quite easily. Its color is yellowish-white, streaked with grayish-red areas, the pyra-

mids being somewhat darker than the surrounding tissue, and well defined. There is an increase in the width of the cortical portion. Microscopic examination reveals marked fatty and granular degeneration of the epithelial cells of the glomeruli and remaining portions of the tubule, the lumina of the tubules in many places being filled with the desquamated epithelial cells. The interstitial portion in some parts reveals inflammatory change, polynuclear leukocytes, red blood-cells, and numerous round cells appearing in scattered areas. The marked fatty degeneration and anemia give the kidney its characteristic color and cause its loss of consistency.

Pathology of Chronic Hemorrhagic Kidney.—The gross appearance of this variety of chronic nephritis closely resembles acute diffuse nephritis; it differs, however, in the fact that the kidney is *very tough.* It is somewhat mottled, there being areas of dark-red and of purplish color intermingling, thus causing the mottled appearance. The capsule begins to show adherence to the kidney substance at various points. The cortical portion is also increased in width, but here and there shows contraction, due to the interstitial alteration. This form of nephritis shows more marked interstitial change than the large white kidney, also areas of hemorrhage intermingled with the other characteristics of chronic parenchymatous nephritis. The form previously described is often with difficulty differentiated from this variety, as the interstitial changes appear in both. The cicatricial tissue may be so marked as to cause atrophy of some of the glomeruli and uriniferous tubules.

Pathology of the Mottled or Secondary Cirrhotic Kidney.—As the process continues, and connective tissue increases, contraction results, so that the kidney now becomes of about normal size. The fibrous capsule becomes thickened at points and quite firmly adherent to the kidney substance, the surface of the organ being slightly irregular and mottled, the darker areas being due to the interstitial change and the lighter ones to the fatty and anemic changes which have previously been described. The organ is quite tough, and narrowing of the cortex becomes apparent at many parts. The cut surface is streaked or mottled. This form of nephritis is quite common, the interstitial and parenchymatous changes proceeding hand in hand. It closely resembles the kidney of chronic interstitial nephritis, and may be said to form the bridge between chronic parenchymatous and chronic interstitial nephritis. From the clinical standpoint, it is known that cessation or abatement of the

symptoms may occur in this variety. However, the destroyed epithelium, which has been replaced by fibrous connective tissue, never regains its normal condition.

General edema and dropsy of the serous cavities is noted in all three of the chronic forms of parenchymatous nephritis; however, hypertrophy of the left ventricle of the heart is most pronounced in the form last described (secondary cirrhotic kidney). Upon postmortem examination, edema is found in many organs, such as the brain and membranes, but there is edema particularly of the lungs. Sclerotic arterial changes are especially likely to occur with the mottled or secondary cirrhotic kidney.

Symptoms of Large White Kidney.—This is a form developed from the acute variety, in those in whom the disease has existed for months, and in whom chronic parenchymatous changes have formed. The urine, which was scanty and high-colored, becomes more copious, often quite abundant in amount. The albuminuria, dropsy, and anemia, which have never entirely disappeared, become more prominent again, and often resist treatment. The characteristic *renal facies* is present. There is marked edema of the feet. The urine is of high specific gravity, and loaded with albumin, twenty grams often being passed in twenty-four hours. Many of the symptoms encountered in the acute form may occur in this variety.

Symptoms of Hemorrhagic Kidney.—The symptoms are similar to those just described, the obstinate dropsy and the hemorrhagic urine being most characteristic. The urine may even increase to the normal amount, yet the dropsy does not entirely disappear. There is slight hypertrophy of the left ventricle, and there may be changes in the walls of the vessels. By some writers (Rosenstein and Aufrecht) it is believed that this is a separate form of renal disease; most observers hold that it is an acute exacerbation of the chronic variety. It is, however, a very rare form of chronic renal disease.

Symptoms of the Mottled or Cirrhotic Kidney.—In this variety the disease is chronic from the onset. The first symptom noted is the dropsy, which, as a rule, is moderate at first, appearing particularly in the eyelids and at the ankles. There is marked anemia, the patient complaining of fatigue, dyspnea, headache, and palpitation upon slight exertion. This variety, under appropriate treatment, may apparently recover; relapses, however, are frequent, the disease continuing for years, until finally some complication closes the scene.

CHEMIC AND MICROSCOPIC CONDITION OF THE URINE IN TYPICAL CASES OF THE THREE VARIETIES OF KIDNEYS SEEN IN CHRONIC DIFFUSE PARENCHYMATOUS NEPHRITIS.

CHEMIC.

URINE.	LARGE WHITE KIDNEY.	MOTTLED OR SECONDARY CIRRHOTIC KIDNEY.	CHRONIC HEMORRHAGIC KIDNEY.
Amount	Diminished considerably.	Normal or above.	Diminished.
Color	Yellow; yellowish-red.	Light yellow; turbid.	Reddish-yellow.
Specific gravity . .	Above normal.	Normal or below.	Above normal.
Reaction	Acid.	Acid.	Acid.
Sediment	Present; often abundant.	Present.	Present.
Urea	Diminished.	Diminished.	Diminished.
Albumin	Abundant, $\frac{1}{10}$ to 1% or more.	Less than large white kidney.	Abundant.
MICROSCOPIC.			
Crystals	Urates.	Rarely found.	Rare.
Casts	Granular, hyaline, and fatty casts.	Granular and hyaline casts especially.	Granular, hyaline, and sometimes blood-casts.
Cells	Degenerated epithelial cells, especially fatty; leukocytes.	Degenerated epithelial cells; leukocytes; rarely red blood-corpuscles.	Epithelial cells, red blood-corpuscles, and leukocytes.

The Urine.—The specific gravity varies from 1015 to 1040. It is light yellow in color, often cloudy, and acid in reaction. The amount of albumin contained in the urine is large, from 1% to 5%, more albumin being passed after exercise than after rest. The urea is diminished in amount. Microscopic examination reveals granular large and small hyaline casts, and occasionally epithelial and fatty casts. There are also observed granular debris, leukocytes, and degenerated epithelial cells.

Dropsy.—This is a most constant symptom, affecting the face, the extremities, the scrotum, and the serous cavities.

Gastric Symptoms.—These are common. Nausea and vomiting often occur, the appetite is lost, the tongue is coated, and there is constipation alternating with diarrhea.

Blood.—An anemia of the chlorotic type often occurs. The

erythrocytes may be diminished to 800,000 per cubic millimeter. The tendency to hemorrhage is marked.

Changes in the Circulatory System.—Some changes take place in the heart, such as hypertrophy with dilatation, especially with the secondary cirrhotic kidney. The second aortic sound is often accentuated, and hemic murmurs may be heard over the base of the heart.

Eye Symptoms.—Often there is dimness of vision, and specks and mists float before the eyes. Albuminuric retinitis occurs, but is not so common as in chronic interstitial nephritis.

Complications.—Pleurisy, pneumonia, pericarditis, meningitis, erysipelas, gangrene of the skin, and edema of the lungs are of common occurrence. If cardiac hypertrophy becomes marked, apoplexy is liable to result. Uremic phenomena are frequent.

Differential Diagnosis.[1]—

Large White Kidney.	*Chronic Hemorrhagic Kidney.*	*Mottled or Secondary Cirrhotic Kidney.*
Edema extensive and tenacious; anemia marked.	Edema excessive and obstinate; anemia marked.	Edema varies, often nearly disappears; anemia marked.
Heart shows no hypertrophy.	Heart slightly hypertrophied.	Heart hypertrophied; often dilated.
Urine of highest specific gravity; largest amount of albumin; microscopic elements show fatty changes.	Urine has abundant albumin; red corpuscles in large amount and constant.	Albumin in urine varies; the specific gravity is lower; casts often disappear.

Prognosis.—Complete recovery is extremely rare. The disease may be delayed for years, but even the mildest cases may terminate in uremia. The great liability to complications should always be borne in mind.

Treatment.—The patient should be protected by warm clothing, flannels being worn during the entire year. If possible, the patient should live in a warm, dry climate, remaining in the sun the greater part of the day. The functions of the skin must be maintained. Warm baths followed by friction and rubbing of the skin are useful. The diet is most important, milk being the best food. Kumiss and buttermilk may also be given, or milk and Vichy water. In mild cases the following articles may be allowed sparingly: Fish, white meats (such as veal and white meat of chicken), green vegetables, and small quantities of bread. Alcohol and malt liquors

[1] Modified from Loomis-Thompson, "American System of Practical Medicine."

should be *strictly prohibited.* Water must be taken in large amounts. Tobacco, if used at all, should be indulged in sparingly. Moderate exercise, avoiding fatigue, is of advantage. Bicycling is to be strictly forbidden.

The functions of the body require careful attention. Constipation can best be treated by the administration of broken doses of calomel from time to time, followed by a saline. When the amount of urine has been markedly decreased, the use of digitalis is found beneficial, as is also Basham's mixture. One-drop doses of the tincture of cantharides three times a day in Basham's mixture will be found of advantage when the amount of the urine has become small. Complications must be treated upon general principles.

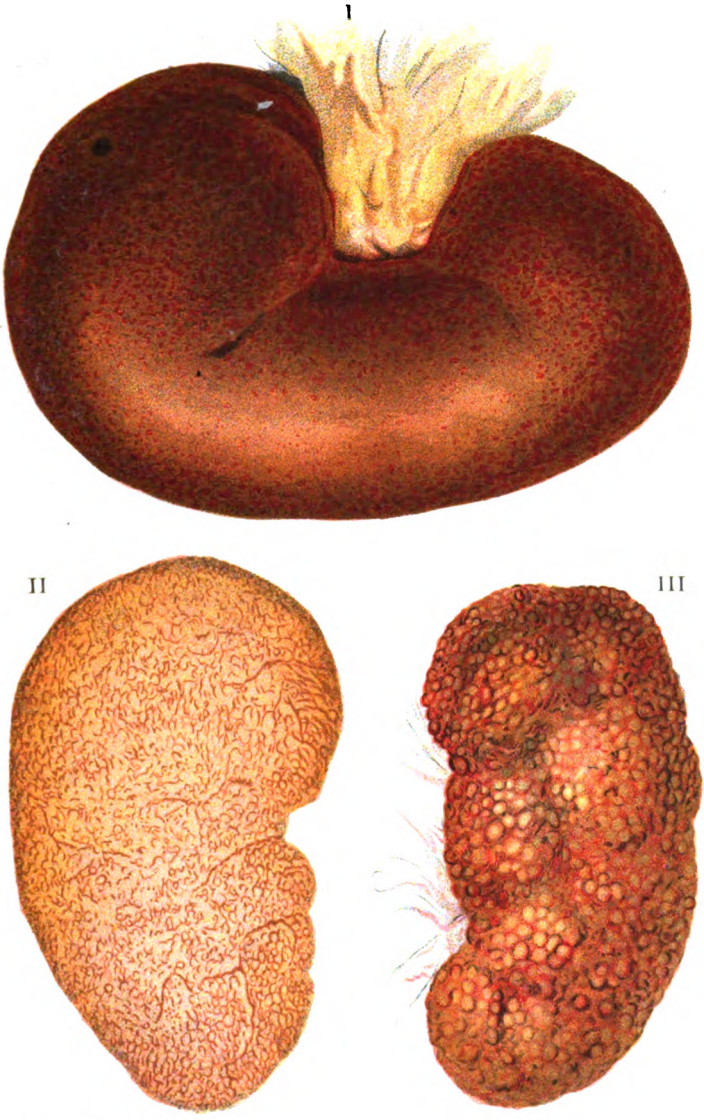
CHRONIC INTERSTITIAL NEPHRITIS.

Synonyms.—Chronic contracted kidney; granular kidney; cirrhosis of the kidney; gouty kidney; small red kidney.

Etiology.—The disease occurs most frequently in males between the ages of forty and sixty. It arises particularly in the lithemic or gouty diathesis, and in those subjects who early show a tendency to fibroid changes in the arterial system. Alcohol is one of the principal predisposing causes. Partaking of rich food, with very little exercise, is also a causative factor. Gout and syphilis are important causes.

Pathology.—In this form of nephritis the kidney is usually markedly reduced in size, sometimes weighing as little as fifty grams; however, when this change occurs, both kidneys are not liable to be affected to the same extent. When the kidney is very small, the perirenal fat is often extensive. If the process be of short duration or of slow development, it may be of almost normal size, so that in a measure it may quite closely resemble the secondary cirrhotic kidney. The surface of the organ becomes granular, this varying from a finely granular to a somewhat hobnail appearance. The fibrous capsule is drawn down in many points, giving rise to a granular appearance, is firmly adherent to the kidney substance, and anastomosis between the blood-vessels of the cortex and the capsule may take place. The color of the surface is reddish or reddish-gray, sometimes red-brown, intermingled with lighter reddish-yellow areas. Frequently *retention cysts* are visible under the fibrous capsule, which vary in size from a pinhead to a pea, or occasionally they may be larger. These are filled with

Plate VIII.



I. Acute hemorrhagic nephritis.
II. Chronic parenchymatous nephritis.
III. Chronic interstitial nephritis. (From Rosenstein, "Pathologie und Therapie der Nierenkrankheiten.")

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a clear fluid, rarely turbid or purulent. The kidney appears tough, and upon section the knife meets with much resistance (leathery consistency). On viewing the cut surface it will be found that the entire width of the kidney is decreased, but the cortical portion suffers most, so that it is narrowed, and occasionally forms but a faint rim. The marked narrowing of this part of the kidney is due to the fact that it is the most delicate, while the medullary portion contains a stronger supporting structure, so that when the newly formed connective tissue in the cortex contracts, the cortex suffers most. The pelvis of the kidney occasionally may be larger than normal on account of the contraction of the organ, but it is always so on account of the disproportion in size. In chronic nephritis due to gout and lead-poisoning it not infrequently happens that infarcts are found that contain uric acid deposits or calcareous infiltration. *Microscopic examination* reveals the interstitial change as being more marked at certain points, but particularly localized in the cortex. The fibrous connective tissue is found to separate many of the tubules, and there also appears here and there marked cellular infiltration of round cells and leukocytes. The epithelial cells lining the tubules show granular and fatty degeneration in limited areas, but the greater number of the epithelial cells appear normal, and in stained preparations the nucleus of these cells has its normal selective power for the basic stains. The epithelial cells of the Malpighian bodies may also show similar changes. As the new-formed fibrous tissue contracts, some of the tubules and glomeruli atrophy, many of them being completely replaced by areas (or whorls) of sclerotic tissue. When the contraction takes place around the tubule at some distance from the Malpighian body, and blocks up the lumen, a cyst forms, which has previously been described, and this appears under the microscope as a large space. The arteries are also thickened, the intima, the media, or the adventitia showing sclerotic changes; and often the outer coat is continuous with the surrounding new-formed fibrous tissue. It will also be noted that in some areas the glomeruli and tubules are hypertrophied, they being larger than normal, and the lumina of the tubules being well marked. The veins rarely show any change, but occasionally the outer coat is thickened. From the narrowing of the lumina of the arteries anastomotic changes arise. The heart in this form of nephritis shows *marked hypertrophy;* this in the greater number of cases is

chiefly limited to the left ventricle, but may involve the entire organ. It is also frequently associated with dilatation. Arteriosclerosis of many of the blood-vessels of the body frequently causes chronic interstitial nephritis. Congenital narrowing of the aorta in young chlorotic individuals has been noted, which subsequently gives rise to chronic contracted kidney. A contracted kidney is found on the postmortem table which during life may not have given rise to characteristic symptoms.

Symptoms.—The symptoms may be so ill defined as to escape detection. As a rule, muscular weakness and lassitude develop. There is gradual loss of appetite, dyspepsia sometimes appearing. Headache, pain in the neck and back, irritability of temper, loss of memory, and sleeplessness are all early symptoms. The amount of urine is greatly increased, from 3000 to 6000 c.c. being passed in a day; it is clear, acid in reaction, and of a pale, slightly greenish color. The specific gravity is low—from 1004 to 1012. Albumin, if present at all, is found in very small amounts; it may be absent altogether. The amount of urea eliminated is diminished. Some few hyaline casts may be discovered upon microscopic examination. The important characteristic symptoms relate to the circulatory system, consisting of marked hypertrophy of the left ventricle. The arterial tension is increased. The pulse is firm. There is an accentuated ringing sound at the aortic cartilage; the apex-beat is forcible and heaving. As long as compensation is maintained no symptoms are developed; however, dilatation and cardiac insufficiency soon arise. There is shortness of breath upon slight exertion, and palpitation, and gradually the symptoms of edema of the lungs appear. The patient is pale, and the eyelids may be slightly swollen. The skin of the body is dry, there being but slight tendency to sweating. Marked anemia of the chlorotic type is present. Edema, as a rule, is absent; when it occurs, it is due to secondary parenchymatous changes or failure of compensation. The eye symptoms are important, albuminuric retinitis being common in this condition.

Complications.—Cerebral hemorrhage is a frequent complication. Inflammation of the serous membranes is common. Peritonitis, pericarditis, pleurisy, pleurisy with effusion, chronic pericarditis, bronchopneumonia, lobar pneumonia, neuroretinitis and retinal hemorrhages, and edema of the glottis are all complications.

Prognosis.—When the disease has once become established, there is no likelihood of a cure. The patient may live for a number of years, the average duration of life being from three to five years.

Treatment.—The treatment in regard to general hygiene, diet, and so on, is the same as in other forms of renal disease. Diuretics are not indicated. Iodid of potassium is a valuable drug, continued for weeks at a time. For the palpitation and cardiac difficulty nitroglycerin is of use. Complications must be treated as they arise.

AMYLOID DISEASE OF THE KIDNEY.

This is a disease of the kidney often associated with chronic parenchymatous nephritis; it may, however, exist independently. When it is present, other organs besides the kidney are frequently affected.

Etiology.—The condition occurs most often in cachectic individuals. It may arise at any period of life, but most frequently between the ages of twenty and thirty, and it is found in both sexes. Pulmonary tuberculosis, empyema, bronchiectasis, abscess formations of various tissues, especially of bone, chronic malaria, and syphilis are all predisposing causes.

Pathology.—The organ is usually increased in size, sometimes being twice that of the normal kidney, and closely resembling the large white kidney. The fibrous capsule strips quite readily, the kidney being of a dull yellow color. It is of a tough, bacony consistency. On section, the cortical portion is increased in width, the Malpighian bodies often standing out prominently as small, white, glistening points. When treated with Lugol's solution, the characteristic amyloid reaction is obtained, the Malpighian bodies and the blood-vessels particularly being stained a mahogany brown, while the surrounding kidney structure is a light yellow. The reaction is better illustrated with some of the anilin dyes. (For Amyloid Reaction, see p. 520.) Cirrhotic changes not infrequently are found associated with this condition. The organ, under such circumstances, is of about normal size, very tough, and upon microscopic examination reveals the increased fibrous connective tissue. Parenchymatous changes are also associated.

Symptoms.—The disease is always preceded by some chronic process, very frequently a suppurative one. The

patient becomes weaker, and dyspnea is more marked, being increased upon exertion; he becomes very pale, and the complexion has a waxy cast. The urine passed is increased in quantity, micturition being more frequent, the patient being compelled to get up at night and pass urine. Upon examination of the abdomen, the liver and spleen are found to be enlarged. An examination of the urine shows that it is light in color, often straw-colored, and is of low specific gravity—1005 to 1015. In the scanty sediment are found large hyaline, small granular, and broad waxy casts, with fatty globules adhering to them. Besides these there are found microscopically leukocytes and fatty epithelium. Albumin is almost always present; exceedingly rarely is it absent. The amount of urea excreted is below normal. Dropsy is usually present, and it often shows itself as anasarca, the subcutaneous tissues and serous cavities being infiltrated. The edema, when once it appears, is persistent. The gastric disturbances consist in nausea, anorexia, severe vomiting, and diarrhea. Among the circulatory symptoms, hypertrophy of the heart is found to be absent. Only in rare instances do changes in the heart-wall occur. The temperature, as a rule, is normal.

Prognosis.—The prognosis is unfavorable, the duration of the disease being variable.

Treatment.—General tonic treatment by arsenic, iron, iodid of potassium, and cod-liver oil are of use. The same hygienic regulations, diet, and exercise should be insisted upon as in other forms of renal disease.

SUPPURATIVE NEPHRITIS.

Synonyms.—Abscess of the kidney; acute interstitial nephritis.

This disease was known to Hippocrates, and Galen and many of the oldest authors have described it.

Etiology.—This condition is clinically always of bacteriologic origin. It may be due to chemic substances which induce suppuration. Experimentally it is known that turpentine may produce suppuration. The most important micro-organisms which have been found as causative factors are the staphylococcus pyogenes aureus, the streptococcus pyogenes, more rarely the bacterium coli communis, also the gonococcus, the tubercle bacillus, the diplococcus of pneumonia, the bacillus typhosus, and the ray fungus. There are four ways in

which the organism may reach the kidney: (1) Externally through a perforation (trauma); (2) from infection by extension through the perirenal tissues; (3) from the urinary passages, traveling upward to the kidneys through the urethra, bladder, prostate, and ureters; (4) through the blood, as in pyemia. The cases arising from trauma occur particularly in young individuals. Those arising from the second cause are most common in the female sex, in which perimetritis plays the chief rôle. The third arises most frequently in the male sex, after the fortieth year; and under the fourth heading, the affection occurs at any age, and may be met with in the newborn.

Pathology.—When resulting from trauma, the suppurative process may be slight, or it may be so extensive as to involve the whole organ, which exists as a pus sac.

Pus may find its way through the ureters into the bladder, or through a fistula to the external part of the body, or to other organs, such as the bowel, peritoneum, liver, bronchi, or the lung structure itself; and, lastly, pyemia may follow. When arising by contiguity and continuity of structure, the picture is similar to that just described; except that the kidney substance is not excoriated, as is so frequently the case, and also that hemorrhages are not apparent. When the infection extends from the urinary passages, they are identical with those which will be described under pyelonephritis. When the infection is brought to the kidney through the blood channels, both organs are involved; the kidney is somewhat irregular in outline, and the surface shows yellowish areas surrounded by a hemorrhagic zone. When the capsule is removed, it is adherent in some parts. On section, numerous abscesses are exposed, varying in size from a pinhead to a pea, rarely larger. The pus is yellowish or yellowish-red, the latter depending upon blood being admixed. Upon microscopic examination the characteristics of acute inflammation leading to suppuration are noted. If the suppuration be chronic and long-continued, thickening by fibrous tissue is frequently noted around the abscess. Sometimes it is found that cicatricial tissue has replaced a small abscess. This is most likely to occur from traumatic influences. Compensatory hyperemia of the healthy portion of the organ or of its fellow upon the opposite side is common. In the pyemic variety these changes are not always observed, as the process is very rapid.

Symptoms.—Abscesses of the kidney may exist without giving rise to characteristic symptoms, or the symptoms are masked by those of the primary disease. The most characteristic symptoms occur in the traumatic variety, in which the remains of the injury may be seen in the renal region, also tenderness upon pressure. Symptoms of shock, and either hematuria or anuria, chills which may be repeated, followed by fever, may occur at the onset, or may occur in a few days, when abscess formation has taken place. If the subsequent course of the injury has not been severe enough to cause death, it may be that the pus has emptied itself through the pelvis of the kidney and has been discharged with the urine. In such cases the fever declines day by day until the discharge of pus has ceased entirely. The pus may find an artificial opening, forming a fistulous tract either internally or externally. If this latter condition arise, the fever is apt to be prolonged and of a pyemic character unless the pus should find its way into the peritoneal cavity or erode a large blood-vessel, leading to speedy death.

In cases in which abscesses of the kidney have occurred from continuity of structure, the symptoms are less marked. There may be fever, pain in the abdomen and back, and difficulty in urination. A tumor may be found in the renal region, which is painful upon palpation. The appearance of pus in the urine in such cases does not necessarily point to abscess of the kidney, as the pus may have found its way into the urinary passages without affecting the kidney. Only in those cases in which, with the appearance of pus in the urine, a tumor in the renal region has diminished in size or entirely disappeared, is a diagnosis of renal abscess permissible. The further progress of the affection is the same as in the traumatic variety, except that the prognosis is not so apt to be favorable. In both forms the urine may appear perfectly normal as regards the quantity, color, etc. Especially is this the case when only one kidney is affected. Occasionally, however, the urine shows marked changes, particularly in cases in which both kidneys are affected by a purulent process. In such cases the quantity of urine is usually normal. The reaction is acid, or, from the presence of considerable quantities of pus, it may be either neutral or even alkaline, and its appearance turbid, and the decomposing purulent material in the urine is apt to be ammoniacal. Albumin, as a rule, is present only in small

amounts, except where the purulent process may have produced a parenchymatous nephritis. In such cases the urine will show all the characteristics of parenchymatous nephritis, large quantities of albumin, and various forms of tube casts. Occasionally, small particles of kidney substance, which have become loosened in the purulent process, may be passed with the urine. When the amount of urine has been markedly diminished, the danger of uremia must be thought of, although this is an exceedingly rare sequence of suppurative nephritis.

The symptoms due to suppurative nephritis occurring from infection of the urinary passages will be mentioned under pyelonephritis. The symptoms of suppurative nephritis due to pyemic and septopyemic conditions are those of the general process, and are not particularly referable to the kidney. There may be marked changes in the urine. If large numbers of micro-organisms be found in the urine, or anuria occur, this condition must be suspected.

Diagnosis.—Suppurative nephritis due to pyemic conditions can not be diagnosticated with certainty; it should be suspected when in the course of the pyemic process albuminuria and pus in the urine suddenly show themselves without the symptoms of parenchymatous nephritis. The conditions which are most important in leading to a diagnosis are a preceding history of trauma or of a source of infection, and the symptoms which particularly point to this condition are pain in the renal region, fever of the pyemic type, enlargement or tumor in the region of a kidney, and the presence of pus in the urine. *Of all these symptoms, the appearance of a tumor in the renal region is the most important.*

Prognosis.—The primary traumatic cases give a more favorable prognosis than the other varieties of suppurative nephritis. In the pyemic form the prognosis is necessarily unfavorable.

Treatment.—If pus can positively be diagnosticated in the kidney the indication to release the pus or entirely to remove the kidney becomes imperative; which method is more likely to be successful, must be left to the judgment of a competent surgeon. If for any reason removal of the pus is not practicable, the condition must be treated symptomatically. The pain should be lessened, attempts should be made to reduce the fever, and the general constitution of the patient maintained by a concentrated nutritious liquid diet, stimulants, and tonics.

PERINEPHRITIC ABSCESS.

This condition may arise from suppurative diseases of the spine, the pus finding its way into the tissues surrounding the kidney. Infection may also arise from suppuration of the bowel, not uncommonly from disease of the vermiform appendix, from abscess of the liver, from an empyema, and, lastly, from abscess of the kidney itself.

Symptoms.—Pain in the lumbar region, of a dull, aching character, and occasionally referred to the hip-joint or thigh, is a prominent symptom. The thigh of the affected side is partially flexed, and the testicle may be retracted. Upon examination it will be found that the pain is aggravated upon pressure. There may be bulging of the loin, and occasionally edema and fluctuation are noticed in this region. A large tumor is sometimes distinctly palpable. Fever of a septic type is usually encountered.

Treatment.—The treatment is surgical. Early evacuation of the pus is indicated. Tonics and stimulants are necessary to support the patient.

FATTY DEGENERATION AND FATTY INFILTRATION OF THE KIDNEY.

It is improbable that fatty infiltration ever occurs in the kidney in the human subject; however, fat is sometimes eliminated, and passes through the vessels of the glomeruli into the tubules. Fatty degeneration of the kidney, affecting the epithelial cells, is so constant a feature of chronic parenchymatous nephritis that it might be considered with that condition; it results from severe anemia, particularly progressive pernicious anemia, and from the administration of phosphorus, arsenic, antimonium, cantharides, chloroform, carbon dioxid, chromic acid, aloin, and iodoform; and, also, some one of the infectious processes may give rise to this condition.

PYELITIS AND PYELONEPHRITIS.

Etiology.—The most frequent cause of this disease is mechanical irritation from foreign bodies, particularly renal calculi, the stone often being sharp, causing destruction of the mucous membrane; however, finer calculi (gravel) may also

produce mechanical irritation. This condition results more rarely from tumors, such as carcinoma and tubercle, rarely also from parasites, from extension upward and from a cystitis. The micro-organisms that most frequently give rise to this condition are the gonococcus and the bacillus coli communis. Such irritants as turpentine, cubebs, copaiba, oil of sandal-wood, cantharides, and mustard have a special action upon the parenchymatous structure of the kidney and the mucous membrane of the pelvis, and may give rise to pyelonephritis. In a similar way, it may occur in the infectious diseases, especially in smallpox, dysentery, typhus, and cholera. It may also arise from congestion of the organ, as occurs in valvular heart disease, and from displacement of the kidney, the ureter being twisted or pressed upon. The condition may arise from trauma, also from cold and exposure, especially when this affects the abdomen. Pyelitis sometimes follows various inflammatory conditions of the kidney. Inflammation of the tissues surrounding the pelvis of the kidney, such as a perinephritic abscess, may give rise to pyelitis. It is slightly more common in the male than in the female sex, and is more frequent in adults and in old age. The condition is most frequently unilateral; even when it results from cystitis it is more marked on one side than upon the other.

One of the forms of inflammation met with is the acute catarrhal. The mucous membrane of the pelvis becomes swollen and covered with mucus. The epithelial cells appear granular, many being desquamated, and leukocytes and red blood-cells frequently are found in large numbers, as well as various micro-organisms. In chronic catarrhal inflammation the mucous membrane becomes markedly thickened; the color is brownish-red, and the free surface is covered with thick mucus, or in some cases with purulent material. Occasionally ulceration is noted in the mucosa, and sometimes there are cystic formations. The walls of the pelvis may also reveal sclerotic changes, so that they become thicker and more resistant. A fibrinous exudate may occur upon the mucous membrane from severe irritants, such as cantharides, and in the course of infectious processes, as those of a septic and pyemic nature. When this exudate causes obstruction, or if strictures have formed in the course of chronic interstitial changes, the flow of the urine is hindered; the pelvis, and the kidney, itself may become distended, atrophy of the kidney substance resulting, and the

organ being transformed into a sac containing either watery or purulent material (hydronephrosis or pyonephrosis). If the condition be long continued, interstitial changes result in the kidney. When the urine is dammed back into the kidney, dilatation of the uriniferous tubules results; the epithelial cells become degenerated, and the fluid may extravasate into the interstitial portion, often giving rise to abscesses. These may either be small, or, when they unite, form large purulent areas. The abscesses may be limited by the formation of new-formed connective tissue, and in some instances calcareous infiltration results in the capsule. From suppuration of one kidney amyloid disease may result in the opposite organ. The ureter of the affected side usually shows inflammatory changes similar to those described in the pelvis. Compensatory hypertrophy of the unaffected organ, and in some instances hypertrophy of the heart, result. Pyemia sometimes follows.

Symptoms.—Only that form of pyelonephritis occurring acutely and primarily through irritation from calculi and drugs gives rise to characteristic symptoms, the most important of these being changes in the urine. The urine becomes lessened in amount, contains mucus, pus, and frequently blood, and even crystals, especially of uric acid. If a fibrinous exudate be present, small masses of fibrin may be found in the urine. The microscope will show blood, pus corpuscles, pelvic epithelium, crystals of uric acid, oxalates, masses of fibrin, and occasionally parasites.

Frequent urination is an important symptom. Occasionally, even if one kidney be entirely healthy, anuria may occur, this probably being due to reflex action. In chronic pyelitis the urine is rarely diminished; on the contrary, an increased quantity, amounting to two or even three times the normal, is voided. The specific gravity is normal, the reaction acid, and only in cases in which large quantities of pus are present will it be neutral or alkaline; it is then apt to be turbid in appearance, owing to the intermingling of mucus or pus. A change in the character of the urine voided is apt to occur in the course of chronic pyelitis, especially if the flow of the urine from the diseased kidney be hindered by a calculus, by clotted blood, or by a plug of mucus, so that the urine coming from the sound kidney will appear clear and perfectly normal. Pain in the renal region is a common symptom of acute pyelitis. As a rule, this is present in chronic pyelitis. Enlargement of the kidney may take place, as has been indicated

in the pathology, from a hydronephrosis or a pyonephrosis. Some degree of fever is always present in acute pyelitis ; it is moderate in height, and must not be confounded with the so-called urethral fever, which is usually remittent or intermittent in character.

Prognosis.—In acute pyelitis the prognosis is favorable in the main, especially if it be due to renal calculi, or to such irritants as cantharides, or to one of the infectious fevers, in which the etiologic factor is apt to subside. In chronic pyelitis, or in cases in which suppuration of the kidney develops, the prognosis is not so favorable, this being especially true if accompanied by amyloid disease.

Treatment.—The patient must lie quietly in bed, protected from cold. The patient should be given a bland diet and alkaline mineral waters. Free application of heat to the renal region may be used. Warm baths are grateful to the patient. When pain occurs, opium in some form, as opium hypodermically or opium suppositories, is useful. In chronic pyelitis astringents are indicated, such as acetate of lead, uva ursi, etc. Salol and methylene-blue are sometimes found of benefit. Surgical interference is necessary if hydronephrosis or pyelonephrosis is present.

HYDRONEPHROSIS.

Definition.—Obstruction to some part of the ureters, bladder, or urethra gives rise to dilatation of the pelves and tubules of the kidney, from the accumulation of the urine.

Etiology.—Twisting of the ureter, as is met with in floating kidney, may give rise to the condition, or the ureter may be obstructed by an accumulation of parasites. It may be the result of congenital narrowing of the ureter or urethra. Pressure upon the ureter may result from fibrous bands or tumors. Calculi, tumors, and strictures may occlude the lumen. Enlargement of the prostate in many cases gives rise to the obstruction of the ureter. The accumulation of fluid in some instances causes a very large swelling, which may be mistaken for ascites. Compensatory hypertrophy of the opposite organ may occur, and hypertrophy of the heart is rarely encountered.

Sometimes, when the obstruction is due to calculi or to the twisting of the ureter, an intermittent hydronephrosis is produced, the distention occurring paroxysmally. When the en-

largement is small, it is often not recognized until the autopsy is made.

Upon examination of the patient, a swelling is met with in the renal region, but, as before stated, when extensive, or when occurring in the floating kidney or the "horseshoe" kidney, it may occur in any part of the abdomen, and so may be mistaken for ovarian cyst or ascites. A unilateral is much more frequent than a bilateral hydronephrosis. Upon percussion, flatness is noted over the tumor. The dilatation may become so marked as to cause rupture and the discharge of the fluid. In some instances suppuration follows, and when it is double, uremia may occur. The urine in intermittent hydronephrosis naturally varies greatly in amount, so that polyuria alternating with oliguria may be present. Pain is not a constant symptom, and when it occurs, it is slight, often produced only by pressure. Slight fever may be met with. The examination of the urine shows no characteristic signs of this condition except, perhaps, in the amount voided at intervals. It may contain numerous pus cells and epithelium.

Treatment.—In some instances massage may be practised with favorable results, but the manœuver must always be performed with great care. If hydronephrosis be due to the floating kidney, a pad and a binder properly applied are often of great benefit. Aspiration and drainage are sometimes necessary.

TUMORS OF THE KIDNEY.

BENIGN TUMORS.

Fibroma, lipoma, angioma, lymphoma, which are met with in leukemia, occur; and, according to Rayer, osteoma and chondroma rarely are found. On account of small size, benign tumors do not give rise to symptoms. Adenomata occur in interstitial nephritis; they are usually from the size of a pea to that of a walnut. They are sometimes congenital. Adenomata sometimes occur with carcinomata (adenocarcinoma).

MALIGNANT TUMORS OF THE KIDNEY.

Carcinoma of the Kidney.—This tumor may be met with as a primary or secondary growth. Of all malignant tumors, it occurs in this organ in only about 2% of the cases, including both primary and secondary growths. It is a remarkable fact that carcinoma of the kidney is relatively more frequent in the young than at the age when malignant disease usually

occurs. It is more common in males than in females. Trauma seems to predispose.

Of the primary carcinomata, the encephaloid variety is more common than the scirrhous form. It may affect one or both organs, and seems to be more frequent in the right than in the left kidney. The tumor is commonly of large size, so that in adults it has been found to weigh fifty pounds. When the tumor is secondary, it is nearly always bilateral, and involves almost exclusively the cortical portion, exceedingly rarely the medullary. The tumors vary in size from a pea to a walnut, and resemble the primary growth.

Symptoms.—Primary carcinoma in its onset produces few or no symptoms, so that the nature of the affection can scarcely be suspected. The characteristic symptoms consist of pain in the renal region, hematuria, enlargement of the kidney, and cachexia; however, no particular one of these symptoms is constant. Pain is commonly an early symptom, and lasts throughout the course of the disease. As a rule, it is localized in the renal region, but it may radiate; occasionally it shows itself as a severe neuralgia, or it may be colic-like. Hematuria may arise at any time in the course of the affection; usually, however, it is one of the earlier symptoms. This form is not accompanied by pain. Enlargement in the renal region is the most constant of all symptoms; the growth may produce a tumor which is readily palpable, the size varying in individual cases. As a rule, however, a large-sized tumor is formed in the affected area. If pressure occurs upon the veins, varices and edema of the lower extremities may take place; if the portal vein be pressed upon, ascites occurs. The adjacent lymphatic glands are enlarged, especially in the secondary variety. Cachexia and emaciation are present sooner or later in the course of the affection. Disturbance of digestion, especially diarrhea, and nervous symptoms, which may often be uremic in nature, or due to autointoxication, develop. Fever, when not due to complication, shows itself toward the close of the affection. In some cases death occurs from collapse, with subnormal temperature. The duration of the disease is from a few months to a year or more. In children the course of the affection is shorter.

Prognosis.—The prognosis is grave.

Sarcoma of the Kidney.—Primary sarcoma is less frequent than carcinoma, and also particularly met with in childhood, but may occur at any age, even in the new-born. It occurs

more often in females than in males, the left organ being more frequently involved. Two-thirds of all the cases have occurred in the first ten years of life. The following varieties have been met with : Primary and secondary sarcomata, round-cell and spindle-cell sarcomata, fibrosarcomata, myosarcomata, angiosarcomata, melanosarcomata, and adenosarcomata. Sarcoma of the kidney is commonly a soft tumor.

Symptoms.—Sarcoma can not be clinically differentiated from carcinoma, as the symptoms are practically the same. The point of differentiation between primary sarcoma and primary carcinoma might consist in the fact that sarcomata are somewhat more frequent in children than are carcinomata ; and glandular enlargement is common in carcinomata.

Prognosis.—The prognosis is grave.

Treatment.—The treatment is surgical.

CYSTS OF THE KIDNEY.

These may be congenital or acquired, unilateral or bilateral, varying from a very small cystic mass to large cysts. The most frequent variety are those of small size met with in chronic interstitial nephritis. The formation of these has been described in the pathology of chronic interstitial nephritis. Congenital cysts are almost always bilateral, the entire organ frequently being composed of cysts. Large ones are sometimes encountered. They are more frequent in men than in women, and especially in those beyond middle life. Echinococcus cysts sometimes occur in the kidney but are very rare.

Symptoms.—Cysts may occur without giving rise to any symptoms. The symptoms of chronic interstitial nephritis are commonly met with. Hematuria may occur.

MALFORMATION AND MALPOSITION OF THE KIDNEY.

MALFORMATIONS.

Malformations of the kidney are usually congenital, and when acquired are the result of diseased conditions of the kidney. The most common forms encountered are the lobulated kidneys, supernumerary organs, and fusion of kidneys (either by fibrous tissue or by kidney substance), such as the "horseshoe" kidney. One or both kidneys may be absent, enlarged, or atrophied.

FLOATING KIDNEY.

Synonyms.—Movable kidney; palpable kidney; nephroptosis.

The movable kidney occurs more frequently in women than in men, it being more often met with in the female sex, on account of tight lacing and also as a result of pregnancy. The right organ is more frequently involved. The organ may be displaced by tumors pressing upon it or dragging it down. Heavy lifting and trauma are also etiologic factors. Resorption of the perirenal fat may give rise to the movable kidney. It is frequent in neurasthenics. The organ may be quite freely movable or very slightly so. Three varieties have been described: (1) The *palpable kidney*, the lower border of which can be felt upon deep pressure; (2) the *movable kidney*, in which the upper edge can be palpated on deep inspiration; and (3) the *floating kidney*, the organ being so movable as to reach above Poupart's ligament or quite freely in the abdomen.

Symptoms.—These vary greatly: in some instances no discomfort is experienced by the patient. The common symptoms are those of a dull dragging pain in the abdomen, accompanied by symptoms of neurasthenia or hysteria. Dyspeptic symptoms are common. In some instances attacks of severe abdominal pain, vomiting, chills, and fever, and occasionally collapse are encountered; they are known as *Deitl's crises*, and are proved to be due to twisting of the renal vessels or to strangulation or compression of the kidney. These may be mistaken for renal colic or appendicial colic, and also for the crises which occur in tabes dorsalis. When this occurs, the organ may be distinctly tender upon pressure; however, in the forms of floating kidney without these paroxysmal attacks the organ is rarely if ever tender upon pressure. When the ureter is twisted and stenosis results, hydronephrosis with alternating oliguria and polyuria may develop.

Treatment.—Great relief is obtained by the proper application of a suitable pad and binder. Operative interference consists in fixation of the kidney, and is often necessary.

RENAL CALCULI.

Synonym.—Nephrolithiasis.

Under this term are included all concretions which form outside of the parenchyma of the kidney which have not yet found

their way into the bladder. According to the size, the smallest calculi, which are almost powder-form, are called kidney sand; the larger calculi are called gravel; and a still larger form is known as kidney stone, or renal calculus. These stones are principally composed of uric acid, oxalates, and phosphate and carbonate of calcium (more rarely magnesia and ammonia), and still more rarely of cystin and xanthin, and very exceptionally are composed of indigo.

Etiology.—Renal calculi have been found at all ages; however, they are more common between the thirtieth and the sixtieth years of life, and are found during child-life and adolescence. Stones forming in the bladder occur more frequently early in life, and again after the fiftieth year. The affection is much more common in the male than in the female. Heredity and family tendency are important etiologic factors, especially in the formation of cystin calculi, and to a somewhat less degree in the formation of uric acid stones. Uric acid stones are found particularly in families in which gout is hereditary.

Renal calculi are much more common in some localities than in others; however, no locality may be said to be exempt. The affection is found most frequently upon the continent of Asia. In Africa it is common in Mauritius and Lower Egypt. In Europe it is common in Central Russia, in Holland, in Italy, in Lower Germany, in Hungary, in the eastern districts of England, in Scotland, and in parts of France. In America it is found particularly in Canada. Its more common occurrence in some localities than in others has been attributed to climatic influences, particularly the influence of a damp climate, in the geologic formation of which lime enters largely into the composition of the drinking-water.

For the production of renal calculi two things are necessary. In the first place, a precipitate must form from the urine, and a material must be developed which holds this precipitate together. This production of sediment is independent of the action of the urine. Acid urine never precipitates sulphates and phosphates; on the contrary, uric acid and urates and oxalates are precipitated by acid urine.

The stones vary in size, and, as a rule, the smaller the stones, the more numerous they are. The largest one that has ever been found weighed 30¼ ounces.[1] Stones,

[1] S. Gee, "A Case of Renal Calculi," "Med.-Chir. Transactions," LVII, 1874.

as a rule, involve only one organ, and are more frequently found in the left kidney than in the right. They may be found in the kidney tubules, the calyces, the pelvis, or the ureter. In the latter position they are usually cylindric; when occurring in the calyces, they are irregular in outline. The size and consistency vary, depending upon the position of the stone. When they are made up of urates, which are the most frequent constituents, they are of a yellowish or yellowish-red color, and quite firm, and upon section present a concentric appearance. They are dissolved in caustic soda, and upon the addition of acetic acid the characteristic "whet-stone" crystals are noted. Stones composed of oxalates are next in frequency. These stones are much firmer than those composed of urates. They are of a grayish or grayish-black color, and frequently present a "mulberry" appearance, and are usually combined with urates. They are insoluble in acetic acid, but are soluble in strong mineral acids. On the addition of ammonia the characteristic crystals of oxalates are deposited. Calculi composed of phosphates are rarely found in the kidney, and are deposited when the urine has an alkaline reaction, this condition being met with in pyelitis and pyelonephritis. They are soft, chalky stones, of a grayish or yellowish color, and are dissolved on the addition of organic acids. They frequently contain micro-organisms. Calculi composed of carbonates are found less frequently than the preceding ones. The calculi composed of cystin, xanthin, and indigo are rarely met with, xanthin stones occurring particularly in children. The formation of calculi seems to depend upon inflammatory conditions of the kidney, such as pyelitis, pyelonephritis, parenchymatous and interstitial nephritis; cells, such as desquamated epithelium, frequently being the nucleus. When one kidney is involved, the other organ often reveals compensatory hypertrophy. Sometimes there is hypertrophy of the left ventricle of the heart, or if suppurative inflammation involve one organ, the other kidney frequently becomes involved and amyloid disease sets in.

Symptoms.—These depend largely upon the irritation set up by the stone and upon the signs attending the attempted passage of the calculus. If the stone should lodge in the ureter, the symptoms of hydronephrosis or pyonephrosis occur. If part of the mucous membrane of the urinary passages be injured, hemorrhages and pyelitis result.

The diagnostic phenomena are those which attend *renal*

colic. The calculus may attempt to pass without prodromes. It may follow exercise, such as horseback riding; it may follow errors of diet, or the ingestion of alcoholic stimulants, and so on. The typical attack begins with sudden, sharp, cutting, paroxysmal pain in the renal region, which shows a tendency to radiate, especially toward the bladder, and in the male into the testicle of the affected side, which, through the reflex action of the cremaster muscle, causes a drawing-up of this organ. The pain may radiate toward the inner side of the thigh, not infrequently toward the chest and shoulder. The patient, in an attempt to relax the muscles, flexes the thighs upon the abdomen, and presses the hands against the renal region to relieve the agonizing pain. Many reflex symptoms are apt to occur. There may be chill, followed by active perspiration, frequent desire to micturate, vomiting, and involuntary evacuation of feces and urine. In the severest cases collapse may occur, the pulse becomes small, the extremities grow cold, and in rare instances death has occurred. The urine which is passed during the attack is small in amount, depending upon the grade of stenosis of the ureter and the condition of the other kidney; and not uncommonly there are blood, mucus, and pus present. Anuria has been recorded from reflex action upon the other kidney. These symptoms may also occur if both ureters should be occluded. Anuria, if it should last a few days, gives rise to a fatal uremia.

The duration of an ordinary attack of renal colic is several hours, rarely two days. The pain usually ceases abruptly as the stone enters the bladder. Hematuria occasionally occurs during the course of renal colic. After an attack of renal colic there may be a slight aching in the loin, or the pain may be dull and remain localized for a long period of time if the stone does not pass. If suppuration sets in, the symptoms of pyonephrosis arise.

Diagnosis.—The diagnosis consists in the characteristic pain, its radiation, accompanied by vomiting, chill, fever, sweating, and often urinary changes. The stone may be found in the urine, and of late years the Roentgen rays have been of use in locating the calculus.

Prognosis.—As a rule, the prognosis is favorable, although recurrence is common.

Treatment.—The treatment consists largely of giving the patient relief during an attack of renal colic, which is best accomplished by hypodermics of morphin or by the inhala-

tion of chloroform. Hot baths, and hot fomentations, such as poultices to the loins, are found to be of benefit. The patient should partake freely of fluids. During an attack of renal colic the diet must be light and carefully regulated. The patient should lead a quiet life. The most important drugs for the relief of uric acid calculi are piperazin, urotropin, and salts of lithia. The waters of various mineral springs, particularly Carlsbad, Ems, and Kissingen, are valuable in treatment. When the urine is alkaline, urotropin, in 15- or 20-grain doses once daily, is of benefit. Surgical interference is sometimes necessary.

UREMIA.

Definition.—Uremia is a clinical condition due to acute or chronic diseases of the kidney or of its conducting apparatus (in which stenosis is present). Depending upon whether the symptoms arise rapidly and suddenly or whether they are more insidious, the condition is known as *acute* or *chronic uremia.*

Symptoms.—Sometimes in **acute uremia** the symptoms resemble an epileptic attack, which may be preceded by prodromes, commonly consisting of headache, spots before the eyes, dimness of vision, vertigo, anorexia, nausea and vomiting, and occasionally headache, which may be one-sided, resembling an attack of migraine. Associated symptoms are frequently tinnitus aurium, which is sooner or later followed by complete unconsciousness, coma, with clonic and tonic spasms, which may occur in an extremity, in the face, in the muscles of the neck, the back, or the abdomen. Rarely are these convulsions unilateral. In the course of a few minutes or a quarter of an hour the convulsive movements may cease, the coma continuing, and Cheyne-Stokes respiration may set in, with an increase of the cardiac asthenia. In other instances the coma may cease and the patient may promptly recover, or the condition known as *chronic uremia* may develop. During the height of the attack the pupils are dilated and do not react to light. Cyanosis and relaxation of the sphincter muscles are present. The skin is bathed in perspiration ; rarely it is hot and dry. The pulse-rate is slowed and shows increased arterial tension ; during the convulsive movements it is frequently small and intermittent or remittent. If the temperature be taken after the convulsive movements, an elevation will be ob-

served, which soon declines if no new attack occurs. In cases that prove rapidly fatal the temperature falls to subnormal ranges.

The amount of urine is usually markedly diminished, containing albumin, and, microscopically, casts, renal epithelium, and often red and white blood-cells. Eye phenomena are very commonly observed, such as amaurosis, which may be bilateral, and may be a prodrome of the affection. Albuminuric retinitis is frequently observed.

Chronic Uremia.—All the symptoms which have been described in the acute occur in the chronic variety, with the exception that they are milder. A symptom in chronic uremia is the uriniferous odor, which is often noted by the patient. The skin is usually dry and itchy. The temperature in chronic uremia, in contrast to the acute form, is always normal or subnormal, and if no complications are present, does not give rise to fever. Melancholia and delusional insanity may follow uremic attacks.

Uremia may exist for a long period of time—weeks and sometimes even months. Under these conditions it may be confounded with some of the infectious diseases.

Diagnosis.—In apoplexy, monoplegia or hemiplegia is common, while in uremia they are rarely met with. Alcoholic coma is exceedingly difficult to differentiate from uremia, as in both conditions subnormal temperature is apt to be present, and there is no point of differentiation of the pupil in either case. This one point, however, is of importance—convulsions rarely occur in alcoholic coma, whereas they frequently occur in uremic coma. Very little stress should be laid upon the alcoholic odor of the breath, as it is not at all unlikely that the patient may have partaken of alcohol before the attack, which, indeed, may have been a factor in producing the uremic state. A point of importance lies in the examination of the urine. In the coma from opium narcosis the pupils are contracted, the pulse is slow and regular, the respirations are slow and deep, and convulsions do not occur.

Prognosis.—The prognosis is always grave in uremia; in the acute form extremely so.

Treatment.—The urine should be withdrawn. In young subjects with slow pulse and high arterial tension bleeding is indicated, and should be followed by hypodermoclysis of a normal salt solution. Intravenous injections of a normal salt solution are often indicated. Purging by croton oil and broken

doses of calomel is useful. For the convulsions, chloral, and morphin hypodermically are the most reliable agents. Sweating should be induced by a hot pack or by some similar method. The administration of pilocarpin is accompanied by danger on account of its depressing effect upon the heart.

PART VI.

CONSTITUTIONAL DISEASES.

DIABETES MELLITUS.

Definition.—A constitutional disease characterized by the continuous presence of glucose in the urine.

Synonyms.—Saccharine diabetes; glycosuria; polyuria.

Etiology.—It occurs at all ages and in both sexes. High living is supposed to be a causative factor, but the poor are also subject to it. The excessive use of sugar as a food is by some supposed to produce the condition. Occasionally the disease is hereditary, and may appear in families in which insanity, tuberculosis, and gout prevail. It is more common among the Jews than in other races. Obesity is supposed to favor diabetes. Trauma is an etiologic factor of some importance. Shock, nervous depression, and disease of the brain, especially of the medulla, are causative factors. It occasionally arises in the course of exophthalmic goiter and epilepsy.

Some drugs produce glycosuria, such as chloroform and bromid of potassium. Occasionally glycosuria results after the acute infectious diseases, such as enteric fever, influenza, malaria, and syphilis.

It has been supposed that the disease is of microbic origin, but the question is by no means settled. It is probable that the acute infectious diseases decrease the resistance of the pancreas to the invasions of the micro-organisms which occur so abundantly in the intestines. Pregnancy and parturition occasionally give rise to the condition, and the disease shows itself in the course of some diseases of the liver and pancreas, in malignant diseases of the abdomen, after exposure, and after the ingestion of cold foods.

Pathology.—The body of one dying of diabetes generally shows wasting, and even extreme emaciation. Occasionally,

however, the subcutaneous fat may be quite well preserved. The skin is thin, the hair is scanty, the teeth are defective, and there are often scars left by boils or carbuncles. The brain presents no constant lesion. Often there is congestion and edema, with some thickening of the membrane; less often anemia of these structures is present. Occasionally tumors of the fourth ventricle and of the medulla are found, or softening, sclerosis, and congestion of those parts may be present. Glycogen has been found in large quantities in the medulla and in the sheaths of the vessels of the cortex. The cord presents no lesions that are characteristic.

The heart is often pale and flabby. Pericarditis and endocarditis occur, and fatty degeneration of the muscular fibers is common, and occasionally they are found loaded with fat.

Chemically, the blood contains larger quantities of sugar than in health.

The lungs show changes, congestion, edema, and sometimes tuberculosis. Pleurisy and empyema occasionally occur. The liver is enlarged and soft; it is rarely smaller. Often a certain amount of interstitial hepatitis is present. This form of cirrhosis is sometimes associated with bronzing of the skin. When there is abscess of the liver, there is usually a causal relation between the two conditions. The spleen is small, pale, and flabby. It may, however, be enlarged and congested.

Great attention of late has been directed to the pancreas, and atrophy of this organ has been found in many instances. Various degrees of interstitial inflammation, with the formation of large areas of new tissue, are commonly present. Occasionally cancer of the pancreas has been found associated with diabetes. Cystic disease and pancreatic abscess have also been noticed. The stomach and intestines show no changes that are characteristic.

Some change usually takes place in the kidney, but it is probably secondary to other changes. Commonly there is enlargement and fatty degeneration. Occasionally the cortex is thin and the organ contracted. Hyaline transformation of the epithelium of the tubes is frequently present. The kidney may present all the evidences of chronic diffuse nephritis.

Symptoms.—Two varieties have been described—the acute and the chronic.

Acute diabetes occurs in persons under forty, and most often in children or young adults. The symptoms consist in rapidly oncoming weakness, great thirst, and the passage of

excessively large quantities of urine. The frequency of micturition interferes with sleep. This gives rise to great constitutional depression. The appetite may be increased, even voracious, in spite of which the weight rapidly diminishes. The skin is dry, the lips are parched, the tongue is red and sticky and covered with dark fur, and the secretions from the mouth and bowels are diminished.

There is frequently a complaint of a nauseous, sweet taste in the mouth, and the breath may even have a fruity odor (acetone odor). Muscular strength is impaired, the patient is depressed mentally, and sexual desire is lost. The urine may vary in quantity from five to twenty pints or more, and it contains a large percentage of sugar.

Chronic diabetes is the more frequent form. It occurs in elderly persons of both sexes, and often in those who are decidedly obese. These patients complain of progressive weakness, frequency of micturition, especially at night, and loss of flesh, as well as decrease in sexual desire. Mental depression and disturbance of the digestive organs, with the passage of large quantities of urine, result.

The external appearance is not characteristic, although there may be a peculiar flush upon the cheeks, which may even extend over the entire face. The odor of the breath is often significant.

The nutrition of the skin suffers, so that the epidermis is dry and rough, the nails are brittle, and the hair is thin and dry. The temperature of the body is often subnormal, but at the onset of some acute cases the temperature may reach 103° F. or over. There is often great irritability of temper. Neuralgic affections are common, and the knee-jerks may be either diminished or lost; often, however, they are normal. In women menstruation is often deficient or absent altogether. The appetite is good, commonly excessive. The bowels are usually constipated. The stools may have a fetid odor.

The cardiac impulse is in its normal position, but is often diffused and weak. The pulse may early show high pressure, but in later stages it becomes small and feeble, and is not increased in frequency except when complications occur. A rapid pulse is significant of oncoming coma. The blood contains an excess of sugar and its alkalinity is reduced. The microscopic appearances, however, are usually normal. In advanced cases some anemia may occur. Sugar may be present in the sweat, tears, and saliva, and the body-weight

as a rule, undergoes rapid diminution. The constant presence of sugar in the urine is the important symptom of the disease.

Temporary glycosuria may occur in many surgical conditions, after injuries, in some nervous diseases, and in gout and other general maladies, but the persistent presence of sugar is significant of diabetes mellitus.

Urine.—The urine is greatly increased in quantity, but may vary within wide limits. If there is great diarrhea, the urine may be normal, or even lessened in quantity. The specific gravity is high, varying from 1025 to 1050. As low a specific gravity as 1013 has been observed. The color is a pale greenish-yellow, but it may vary to deep amber. It is usually clear, but may be turbid from the presence of solids. It does not decompose so rapidly as normal urine, and affords a favorable medium for the growth of the yeast fungus. It is always strongly acid in reaction, and uric acid crystals are deposited in considerable amount. The urea is often diminished; the amount of sugar varies greatly. Acetone and diacetic acid are sometimes present in the urine.

Digestive Symptoms.—Hunger is one of the important symptoms. As a rule, impaired digestion does not occur. Occasionally, there are fatty stools, suggestive of pancreatic disease. Diabetic patients are more than ordinarily susceptible to the poison of enteric fever, but the attacks are usually of mild type. In the course of the fever the sugar may disappear from the urine.

Cutaneous Affections.—Boils, carbuncles, and skin eruptions are extremely frequent. Erythema is common. The palms of the hands and soles of the feet may burn intensely, and perspiration may be profuse. Occasionally general sweating occurs. Eczema of the genitals, especially in women, is a distressing symptom. Purpura has also been observed.

There are sometimes small areas of necrosis of the skin at the ankles and on the dorsa of the feet, the disease beginning in a sweat-gland. Cellulitis and gangrene occur in the diabetes of advanced age, and are more frequent in men than in women.

The temperature in the axilla may be subnormal, especially in the morning, whereas in the evening it may be subfebrile. Neuralgias of various kinds are common complications, and sciatica, and especially bilateral sciatica, is suggestive of diabetes. Occasionally symptoms resembling locomotor ataxia occur. They are due to the neuritis affecting the sciatic nerves and their branches.

Affections of the Special Senses.—Diabetes causes impaired vision by weakening the muscles of accommodation; also by diminishing the perception of light in the retina. Cataract, most often of the soft variety, occurs; it may develop very rapidly in young subjects. Inflammation of the retina and atrophy of the optic nerve may also take place.

The senses of smell and taste may be impaired. Deafness from otitis media has occurred.

Diabetic Coma.—The disease may terminate in coma, which occurs at all ages and in both sexes, but is more liable to take place in the young. The exciting causes are supposed to be fatigue, excitement, exposure to cold, and intercurrent acute affections.

The pathology of the condition is obscure. The onset of the coma may be preceded by languor and weakness. The urine may diminish in amount and the specific gravity become lower. An increased rate of respiration is an important symptom. In the majority of cases, however, the condition is ushered in without warning by delirium, often with maniacal excitement, with hurried and deeper respiration, drowsiness soon appearing, which lapses into coma.

The patient breathes from thirty to forty times a minute, with deep, sighing respirations; the pulse is rapid (from 130 to 150 a minute) and feeble. The face is pale, the extremities are cold, and the temperature is subnormal. The secretion of urine is diminished and sometimes suppressed. The bowels are occasionally constipated. Death may be preceded by a rise in temperature, to from 103° F. to 104° F., with convulsions and cyanosis. A peculiar odor of the breath is often present, but is not constant. The urine contains acetone, and is frequently albuminous.

Duration.—In children and young adults the disease is acute, as a rule, and rapidly fatal, lasting from a few weeks to a few months or two years. In elderly persons, especially those who are fat, the disease may continue many years.

Diagnosis.—Diabetes may rarely exist without the presence of many of the symptoms just described. The occurrence of polyuria, with itching, especially of the genitalia, is always suspicious. *The absolute diagnosis consists in the constant presence of sugar in the urine.*

Prognosis.—The disease is always a grave condition. Cases which occur after injury and those which occur acutely are usually rapid in their course. The occurrence of gout and

obesity, with early treatment, are favorable conditions. A marked family tendency to diabetes or to nervous diseases is unfavorable. Death may ensue from any complication, or from pulmonary phthisis, which is a common concomitant. An acute infectious process affecting a diabetic subject is very likely to terminate fatally. Death frequently takes place from gangrene, which may result from trivial causes, such as a slight scratch, or cutting of a corn, etc.

Treatment.—The diet is important; the carbohydrates should be restricted as much as possible. Sugar should not be allowed in any form. The food should consist largely of meats, fish, poultry, eggs, green vegetables and those that are not starchy (such as string-beans, lettuce, water-cress, spinach, young onions, tomatoes, olives, and celery), milk, cream, butter, cheese, and water in large quantities, especially alkaline mineral waters. Climatic treatment, such as the cures at Vichy and at Carlsbad, is of great importance. Exercise and systematic massage are necessary.

Bathing should be allowed if not prolonged and not involving violent exertion. Warm baths and steam baths are especially grateful to the dry, irritated skin. Of drugs, opium and its alkaloids are the most important. Tonics should be given from time to time, and of these, arsenic in some form is perhaps the best. For the diabetic coma, brisk purging is necessary; alkalies, such as potassium citrate in large doses, and copious drafts of water are useful.

Hypodermoclysis of a normal salt solution, inhalation of oxygen, strychnin hypodermically, are also of use in diabetic coma.

DIABETES INSIPIDUS.

Synonyms.—Polyuria; diuresis; polydipsia.

Etiology.—Age is a predisposing cause, the disease occurring most frequently before the thirtieth year. Heredity is important, and diabetes insipidus is more prevalent in the male sex. The disease sometimes follows violent emotions, such as fright, and also follows the ingestion of large quantities of fluids. It may develop during convalescence from acute diseases, such as enteric fever. It occurs occasionally in association with abdominal tumors, tuberculosis, peritonitis, and syphilis. Disorders of the nervous system are supposed to play an important part in the causation, such as injury

to the cerebrospinal axis. It occurs in association with intracranial tumors and in epileptics.

Pathology.—The pathology is obscure. No structural lesion has been found to account for the disease. Diabetes insipidus is believed to be due to a relaxation of the vessels of the kidney.

Symptoms.—The two symptoms which call attention to the disease are the passage of large quantities of urine and excessive thirst. It may begin either rapidly or insidiously. It is often found following injuries, excessive indulgence in water or in alcoholic beverages, excitement, and fright.

When diabetes insipidus is associated with chronic affections of the brain and spinal cord, the onset is often insidious. The quantity of urine may vary in individual cases, and as much as eight or ten quarts may be passed in twenty-four hours. The urine is of light yellow color, and may present a greenish appearance. It is feebly acid or neutral in reaction. The specific gravity is low, rarely reaching as high as 1010. The urine, as a rule, does not contain abnormal ingredients, although albumin is occasionally noted. Thirst is excessive. The secretion of saliva is diminished and the mouth is dry. From the diminished secretion of sweat the skin becomes dry and harsh. The digestion is normal, and gastro-intestinal symptoms rarely occur, although constipation from dryness of the feces may be an important symptom. The temperature is often subnormal. These patients are not subject to cutaneous and pulmonary or other complications, which are so frequent in those suffering from diabetes mellitus. The disease is of long duration; however, some cases run a rapid course.

Diagnosis.—The diagnosis depends upon the large quantity of urine of low specific gravity, the absence of sugar, and the age of the patient.

Differential Diagnosis.—

	Diabetes Insipidus.	*Chronic Interstitial Nephritis.*
Age	Before the thirtieth year.	Middle and advanced age.
Urine	Very large quantities; low specific gravity; rarely albumin and casts.	Large quantities; low specific gravity; traces of albumin and casts may be present.
Vascular changes	Not present.	Hypertrophy of heart; accentuated second aortic sound; arteriosclerosis.
Eye symptoms	Absent.	Common; albuminuric retinitis.
Course	Rarely fatal.	Progressive, always terminating fatally.

Prognosis.—The prognosis as to life is favorable, but the condition is rarely curable. Cases associated with syphilis may recover upon antisyphilitic treatment.

Treatment.—There may be diminution in the quantity of urine and alleviation of thirst under treatment, although the condition itself is incurable. Opium is the most useful drug, but it may interfere with digestion, and there is great danger of the opium habit being acquired. Ergot is occasionally of some use.

CHRONIC RHEUMATISM.

Definition.—Chronic rheumatism is characterized by gradual and permanent changes in the joint structure, producing more or less deformity. The condition often begins insidiously.

Etiology.—The disease is most common in females, and rarely develops before the fortieth year of life. Bad hygiene, exposure to cold and damp, and malnutrition are etiologic factors. Heredity is supposed to have some influence. Occasionally the disease has been seen to follow acute attacks.

Pathology.—Moderate thickening of the joints, with contraction of newly formed fibrous tissue, often ensues. The phalangeal joints of the fingers are commonly affected, as are also the knee-joints. The disease shows slight tendency to symmetry, but this is less striking than in similar conditions, such as arthritic deformities. The joint may show some degree of synovial injection and effusion.

Symptoms.—The symptoms are confined to the joints, and there is no tendency to involve the serous membranes of the heart. The fingers are distorted by contraction of the tendons or ligaments, and deflections, especially of the ulna, are likely to occur. The knees become thickened and stiff, so that the legs can be but partly extended. A crackling may sometimes be heard and felt upon attempting forcibly to extend the part. Redness and edema about the joints are rare. The joint is often much deformed. Atrophy of the muscles may take place about the affected articulation. Constitutional symptoms are mostly absent. Occasionally slight fever ensues from exacerbation of the joint symptoms. The urine is normal; the general health is good; there is occasional anemia and debility.

Prognosis.—The prognosis as to life is good. The condition, however, is incurable.

Treatment.—Local treatment is important, hydrotherapy

yielding good results. The application of hot air in suitable apparatus often gives great relief from pain and stiffness. Massage is also useful. Care should be taken to avoid exposure to cold or wet. Internal remedies are of very little benefit. Occasionally syrup of the iodid of iron and the compound syrup of the hypophosphites are valuable. Cod-liver oil and arsenic as tonics are useful.

GONORRHEAL ARTHRITIS.

Definition.—An infectious disease, characterized by specific urethritis, more rarely conjunctivitis, due to the gonococcus, and by localized inflammation of one or more of the larger joints, especially of the extremities.

Synonyms.—Gonorrheal rheumatism; gonorrheal synovitis.

Etiology.—The disease has no relation to articular rheumatism, and has been erroneously called gonorrheal rheumatism. It is due to the migration of the gonococcus, or its toxins, into the joint. *The etiology, therefore, depends upon the occurrence of a gonococcous infection.*

Pathology.—The affected joint shows the condition common in synovitis. The capsule, synovial membranes, and ligaments are inflamed and thickened. Frequently there is an effusion into the joint, which is rarely purulent. It most often occurs in the knee-joints and ankles. Occasionally the inflammation becomes peri-articular, and extends along the tendon sheaths, invading the periosteum and causing considerable edema. In the chronic form there is often much effusion into the joint. This occurs most commonly in the knee, whereas slight edema appears in the wrist and ankle. As the inflammation subsides, the joint recovers less completely than in rheumatic fever, as fibrous adhesions and thickening commonly remain, producing impairment of motion, and occasionally ankylosis.

Symptoms.—In the acute form the symptoms are mild, and several joints may be involved simultaneously. In the severe, and especially in the chronic, form the disease is often confined to one joint.

The symptoms are usually preceded by a cessation in the flow of pus from the urethra, and the affected joint becomes more and more painful and swollen. The smaller joints are not often involved. The inflammation remains, and does not

show the fleeting character so common in rheumatic fever. The pain is not so intense and redness is not so prominent as in acute articular rheumatism. The pain is constant and intense, often worse at night, preventing all motion of the affected joint. The pain often subsides before the swelling disappears. Fever is moderate, from 101° F. to 103° F. Sweating is not common. Anemia of the chlorotic type is often marked. Anorexia and constipation may occur.

Complications.—Complications are rare. The serous membranes of the heart may become involved, the other viscera escaping. Malignant endocarditis has been seen to follow this affection, gonococci having been found in the endocardium.

Diagnosis.—The diagnosis depends upon the occurrence of arthritis following a specific urethritis or, more rarely, specific conjunctivitis in children. The fact that one joint is affected, and the absence of sweating and of cardiac affections, differentiate the condition from acute rheumatic fever.

Prognosis.—Recovery takes place very slowly, and is prolonged and tedious. There is occasionally permanent stiffness and ankylosis. Relapses are liable to occur with a re-infection.

Treatment.—Increasing doses of the syrup of iodid of iron have proved efficacious in the treatment of this disease. Tonics are necessary on account of the progressive anemia. Cod-liver oil and iron are valuable for this purpose. Local applications are of use, and the joint should not be kept immobile too long on account of the tendency toward ankylosis.

MUSCULAR RHEUMATISM.

Definition.—A diseased condition of the voluntary muscular structures, often involving the surrounding fascia and the periosteum to which the muscles are attached, accompanied by pain and slight swelling.

Synonym.—Myalgia.

Etiology.—The disease is more common in men than in women, owing to the greater exposure and to cold. It often occurs after a wetting from rain, or from sitting in a draft of air, as at an open window. Gouty and lithemic individuals are very subject to attacks of muscular rheumatism. One attack renders the affected person more liable to subsequent suffering upon exposure. As a rule, the disease is acute; it may, however, be subacute or even chronic.

Pathology.—This is by no means understood, some observers claiming that the affection is not due to the muscles, but is due to disease of the sensory nerves; that, in fact, the affection is a neuralgia.

Symptoms.—The prominent symptom of the disease is local pain, without constitutional disturbance, and even in the most aggravated cases fever is entirely absent. The pain varies in severity from a slight dull ache to that of a sharp, stabbing, or lancinating character, and is increased by certain movements. Pressure upon the affected area usually gives prompt relief. The affection lasts from a few hours to some days, the subacute and chronic cases being more prolonged.

Lumbago is a form of myalgia involving the muscles of the back.

Torticollis, or **stiff neck,** affects the muscles of the neck, and is mostly unilateral.

Pleurodynia is a form of myalgia affecting the muscles of the chest.

Treatment.—Rest of the affected parts, hot applications (mustard plasters), hypodermics of morphin, and large doses of the salicylates will, as a rule, prove effective. In lumbago acupuncture is highly recommended. Turkish baths, the hot-air apparatus, and electricity are useful in protracted cases.

RICKETS.

Definition.—Rickets is a constitutional disease characterized anatomically by bone resorption and the formation of new osteoid tissue; clinically, by gastro-intestinal disturbance and nervous symptoms.

Synonyms.—Rachitis; morbus anglicus.

Etiology.—The disease is caused by improper and perverted nutrition, affecting all the structures of the organism. Faulty diet has been given a prominent place in the production of the disease, in causing gastro-intestinal disturbance, accompanied by vomiting and diarrhea. General bad hygiene, improper clothing, lack of fresh air and ventilation, and inherited syphilis are predisposing factors. It is most probable that many of these causes acting together are capable of producing the affection. The disease is most commonly met with in the temperate zone, decreasing in frequency as northern or southern limits are reached. The cold winter months show the larger number of cases. It is equally prevalent in both

sexes. It has not been definitely determined whether the disease is hereditary. It is quite remarkable that the evidences of congenital syphilis are almost entirely absent in rickety children. The principal causative factor is a faulty diet. Farinaceous food without the addition of milk is liable to produce rickets.

Pathology.—Various deformities of bone result in this disease, these consisting principally of bowing or bending of the long bones, such as the femur. The tibia may bend forward; the bones of the head may become altered so that the head becomes box-shaped. Early in the disease the parietal bones become thinned as a result of the bone resorption. Upon palpation of the frontal or parietal bone a peculiar crepitation, resembling that of a parchment-like membrane, is detected, this being called *craniotabes;* later, nodes develop upon the frontal and parietal bones. Deformities also arise in the shape of the chest, so that the characteristic pigeon-breast develops. The sternum becomes thickened; enlargements develop at the ends of the ribs near the costal cartilages, giving rise to what is known as the *rachitic rosary.* In the spine, curvatures are common (scoliosis and kyphosis). The pelvic bones become enlarged and thickened, and the characteristic funnel-shaped pelvis develops. Nodes are also noted at the ends of the long bones. Enlargements of the liver and spleen, chiefly due to an increase in fibrous connective tissue; pronounced anemia, and catarrh of the gastro-intestinal tract are also encountered. Leukocytosis with particular increase in the lymphocytes is common.

Symptoms.—The onset of the disease is insidious. The period of its first dentition is delayed, which often masks the onset of the disease. Gastro-intestinal disturbances give rise to disordered nutrition. The child is peevish and irritable, the temperature frequently being subfebrile. Profuse sweating, restlessness, and sleeplessness, with the fever ranging from 100° F. to 102° F., and a general soreness over all the body, are likely to arise early, as has been described in the pathology. The shape of the head is characteristic; it appears rectangular, the face seeming small in proportion to the skull.

Occasionally, a systolic murmur may be heard by listening over the anterior fontanel. Commonly the spleen is enlarged. The urine contains an excess of the lime-salts. Some degree of anemia is always present; it is commonly of the chlorotic type. The nervous symptoms are usually prominent. There

is restlessness and lack of sleep, and laryngismus stridulus sometimes occurs. Tetany is not uncommon.

Prognosis.—The intercurrent affections which develop in this disease are more to be dreaded than the affection itself, which is rarely fatal. The skeletal deformities do not disappear.

Treatment.—The child should have the best of food, and if the mother can not nurse it, a wet-nurse should be procured. Abundance of fresh air and sunshine are essential. Daily bathing in warm water is necessary. The child should not be encouraged to walk. The drugs that have been recommended are phosphorus, cod-liver oil, and the syrup of the iodid of iron.

ARTHRITIS DEFORMANS.

Definition.—A chronic joint affection, characterized by great progressive deformity, with functional and anatomic changes in the osseous, cartilaginous, and synovial structures.

Synonyms.—Rheumatoid arthritis; rheumatic gout.

Etiology.—The disease bears no relation to either rheumatism or gout. The exciting cause is still unknown, and by many it is supposed to be of nervous origin. The view that the disease is of microbic origin has lately been advanced by Max Schiller, who describes a peculiar form of bacillus obtained from the fluid of the diseased joints. The same microorganism was also found in the blood of the persons affected.

The disease occurs more often in the female sex, in the proportion of five to one. It may appear at any age, but the greater number of cases develop in young adults between the ages of twenty and thirty. In a few instances the disease has appeared to be hereditary. Bad hygiene, mental strain, worry, grief, and care have been cited as etiologic factors. The disease shows an especial tendency to develop among the poorer classes; however, the well-to-do do not escape.

It is sometimes associated with chronic pulmonary tuberculosis. It has followed attacks of influenza, and sometimes arises in sterile women.

Pathology.—The disease involves all the structures of the joint, and the nutrition of the affected part suffers greatly. The lesions usually begin in the cartilages of the articulations, where the circulation is lessened and the friction greatest.

The cartilages of the joints become softened, thinned, and gradually so nearly absorbed that the ends of the bone are in

apposition; proliferative changes ensue at the extremity of the bones, which become thickened; osteophytes develop (which are called Haygarth's nodosities), the ligaments are thickened, and finally partial ankylosis may result. The muscles around the joint become atrophied, and the nerves may show some degree of inflammatory change. In long-standing cases the ends of the bone may undergo atrophy and softening. The small joints of the hands and feet are most frequently involved; however, some of the larger joints are sometimes affected. Deposits of urate of soda are never found around the joints.

Symptoms.—The disease shows great variability in its mode of onset; it is common for the affected person to go to bed apparently healthy, and to awaken in the night with a sharp pain, especially in the joints. There is a sense of tingling and burning in the affected part. Soon a swelling takes place in the affected joint, which comes on exceedingly slowly. The metacarpophalangeal joints are those most commonly affected. In some cases hard nodules develop at the sides of the distal phalanges; these are known as Heberden's nodes. The disease may now be quiescent for months and years; sooner or later, however, other joints are attacked, the swelling and pain in the mean time never entirely subsiding in the joints which were primarily affected; the condition may continue until nearly every joint in the entire body has become affected. At first there may be synovial effusion under the implicated articulation; this is followed by atrophy in the muscles of the involved parts, and occasionally this atrophy is in advance of the joint implication. The nutrition of the parts is affected, especially the nails of the hands and feet. Bursal swellings sometimes occur in the neighborhood of the affected joints, especially upon the dorsal aspects of the wrists. If the disease exists in the joints of the hand or foot, the characteristic fin-like deformity develops. Almost from the beginning of the disease there is increased rapidity of the heart's action; the pulse gradually rises from 90 to 120 or more per minute. This vascular disturbance is not associated with cardiac murmurs; even functional murmurs are absent, as a rule. Generally, during the course of the attack fever does not occur, but with each fresh outbreak a temporary subfebrile range of temperature may be encountered. Pigmentation and glossiness of the skin of the affected area are often observed. This disease is found among children, girls being

more frequently affected than boys, the onset being accompanied by fever. The spleen and lymphatic glands may be enlarged, the joints become stiffened, and the functions limited, accompanied by muscular wasting.

Prognosis.—The prognosis as to cure is unfavorable; however, life may be prolonged for many years. Under proper treatment it is sometimes possible to arrest the progress, but the affected joints can never be restored to their normal functions.

The **treatment** consists in appropriate diet and in careful hygiene. As a rule, meat should be liberally allowed, with a suitable vegetable diet. Exposure to wet and cold must be carefully avoided, and, if possible, the patient should live in a warm, dry climate. Massage, warm baths, and electricity are of decided use. The hot-air treatment has also proved of decided benefit in some cases. The drugs which are of most value in the treatment of this condition are the iodids, particularly the syrup of the iodid of iron, and arsenic. Cod-liver oil is of use as a general tonic. Arsenic should be given in increased doses to the point of tolerance as a tonic.

GOUT.

Definition.—Gout is a constitutional disease, characterized by polyarthritis affecting particularly the small joints and by a deposition of urate of soda in and about the articulations.

Synonym.—Podagra.

Etiology.—The disease occurs most commonly between the ages of thirty-five and fifty, except in the cases marked by strong hereditary tendency, in which the affection may show itself much sooner. It is more common in the male than in the female. Corpulent persons are more predisposed to become gouty, especially those of sedentary habits. Errors in diet are extremely likely to bring on the affection. This applies particularly to the indulgence in red meats and alcoholic beverages. Chronic lead-poisoning also predisposes to attacks of gout. The disease occurs in the well-to-do much more frequently than in the poor; in the latter it is due to overindulgence in malt liquors (poor man's gout).

Pathology.—In gout an increase in urates is found in the blood, which gives rise to supersaturation, and it is believed that this causes the inflammatory changes which develop in

the joints. The involvement first begins in the articular cartilage, thence spreading to the other joint-structures. The characteristic chalk-like deposits (urates) are formed. The joint most commonly involved is the metatarsophalangeal joint of the big toe on the right side. The ankle, knee, small articulations of the wrist, and the hand are also sometimes involved. Chronic interstitial changes are common in many organs, especially in the blood-vessels and the kidneys.

Symptoms of Acute Gout.—The first attack begins suddenly, and usually at night, with intense pain in the great toe, particularly involving the metatarsophalangeal articulation. In the morning it is noticed that the joint is red, swollen, tense, and exquisitely painful. With this there is a slight rise in temperature, the urine being scanty and high-colored and containing sediment (urates). There is great thirst and anorexia. Toward morning the pain subsides somewhat, but in the evening returns with increased severity. The fever continues to rise. Upon the following two or three days the swelling in the joint increases. The pain, however, diminishes, and the fever subsides. The attacks commonly last from a week to ten days. The tenderness and swelling, as a rule, pass away, and the health is restored. Occasionally, it happens that other joints are affected besides those of the great toe, particularly the tarsal and metatarsal joints, and the opposite foot may become involved. In cases of this sort the paroxysm is more prolonged, lasting from two to three weeks; however, recovery from the first attack is usually quite complete. There may now be an interval of about three years or longer before the attack recurs, and after this as the attacks return from year to year they are liable to show marked periodicity, occurring in the spring and fall. As the disease progresses, the larger joints may also become involved, and with each succeeding attack recovery is less complete, so that the joints become permanently enlarged, stiff, and deformed, and the characteristic chalk-stones make their appearance in the hands and the toes, at the knees and the elbows. Large quantities of urates are often found in the urine just after the onset of or following the attack. After the first seizure the patient often has prodromes which warn him of the oncoming of an attack; they most commonly consist of digestive disturbances, such as loss of appetite, pyrosis, flatulency, and irregular action of the bowels. There may be marked nervous disturbances, such as cramps, irritability of temper, neuralgia, depression of spirits,

and cardiac palpitation. These prodromal symptoms disappear suddenly as the seizure comes on.

Symptoms of Irregular Gout.—As a rule, between attacks, especially when the disease has existed but a short time, the patient is free from pain, but this is not invariably so. In chronic cases, especially after repeated attacks, the patient is troubled with irregular pains in the joints, and with many gastric and nervous symptoms, just as in the acute form, except that they are much less severe. This condition has been termed *chronic* or *irregular gout.* Occasionally, it happens that the inflammation in the gouty joints subsides suddenly from exposure to cold or as the result of the application of cold to the joints. This condition is often followed by severe symptoms referable to some internal organ, such as the brain, heart, or stomach. The condition is serious, and often has a fatal issue. It is known as *retrocedent gout.*

Symptoms Referable to the Skin.—Eczema is common, especially of the face, forehead, external ear, neck, and back. As a rule, it is not severe, but is persistent. Psoriasis may occur. Pruritus, local or diffused, which is apt to be troublesome at night, is common. The nails become brittle, and are kept in order with difficulty.

Symptoms Referable to the Eye.—Conjunctivitis and sclerotitis are the most common affections of the eye in this disease. Glaucoma, retinitis, and gouty iritis have been known to occur.

Diagnosis.—The diagnosis of acute gout depends upon the sudden onset with the arthritis, with special exacerbations at night, affecting the small joints, and upon the gastric and nervous symptoms. The disease rarely occurs before the age of thirty or thirty-five.

Prognosis.—The prognosis depends upon the appearance of complications; when these are absent, the prognosis is good. If the kidneys remain sound, the general health is not appreciably lowered. Albumin occurring in the urine is always an unfavorable sign.

Treatment.—Diet and General Hygiene.—As nearly as possible a vegetable diet should be adhered to, as animal food gives rise to uric acid. Water should be partaken of plentifully. Alcoholic and malt liquors, especially the rich, sweet wines, such as port, sherry, and champagne, should particularly be prohibited. Systematic bathing, regular exercise in the open air, avoidance of exposure to cold and dampness, are important.

Treatment of the Paroxysm.—A mild laxative at the onset is useful. If the patient has fever, he should be confined to bed, and the affected joints kept at rest, and a diet of milk and farinaceous articles, with plenty of water, should be insisted on. Bleeding and venesection are contraindicated. The joints should be wrapped in cotton-wool. If the pain become severe at night, opium in some form must be administered. For the attack itself, colchicum is the remedy; from 10 to 20 drops of the wine or tincture may be given two or three times daily. In cases in which colchicum is not well borne, iodid and bromid of potassium are useful. The salicylates or the salts of lithia may be of value.

LITHEMIA.

Synonyms.—American gout; irregular gout.

Etiology.—Excessive indulgence, especially in nitrogenous food, the abuse of alcohol, lack of proper exercise, and heredity have been given as predisposing factors. The neurotic temperament is said to favor the development of the affection.

Pathology.—The pathology is similar to that of gout, except that the joints are not particularly involved, and it is probable that lithemia is due to an accumulation of urates in the blood, as is gout. Da Costa describes lithemia as "a morbid state where the income of nutriment is in excess of the output of waste." Sclerotic changes in the liver, the kidneys, and the blood-vessels are commonly encountered in advanced cases of lithemia.

Symptoms.—The symptoms are frequently vague, but relate particularly to the nervous and digestive systems. The skin and the genito-urinary and circulatory systems, however, also commonly give rise to more or less characteristic phenomena. There is frequently vertigo; tinnitus aurium, insomnia, restlessness, sensations of heat in the soles of the feet, headache, which may be either occipital or general, and occasionally affecting one-half of the head (hemicrania), and hypochondriasis are noted. Gastro-intestinal symptoms are common; the appetite is variable. The tongue is commonly coated; sometimes, however, it is red and dry. There is pyrosis, cardialgia, sensation of weight in the epigastrium, hiccup, occasionally nausea and vomiting, and flatulence, which exists to such an extent that it is distressing to the

patient. Hemorrhoids are common; they may be due to cirrhotic involvement of the liver, which is often tender when pressed upon. The terms "biliousness" and "torpid liver," used so commonly by the older writers, probably in many instances referred to the lithemic state. There is palpitation of the heart, which may be due either to flatulence or to an increased arterial tension. The phenomena of the skin consist in pruritus, eczema, urticaria, and lichen. The urine, as a rule, is of high specific gravity (1025 to 1035), markedly acid, of a dull red color, and on cooling deposits brickdust sediment. Phosphate of lime crystals are also commonly present. Albuminuria is not at all infrequent. Casts may often be found. In another variety of cases neurasthenia develops. The patient becomes anemic. There is great muscular and mental fatigue, with languor.

Prognosis.—The prognosis is good if the patient be able to change his mode of life and to undergo proper treatment. In old cases arteriosclerosis, atrophic cirrhosis of the liver, and chronic renal disease necessarily render the prognosis unfavorable.

Treatment.—The most important elements in treatment are proper diet and hygiene. Meat should be withheld in severe cases, the patient being put upon a diet of cereals and fruit. When improvement takes place, meats may be allowed once a day, preferably the white meats, with fish, white bread, and so on. In the anemic and neurasthenic cases, however, meat is necessary. Large quantities of aerated waters are beneficial. Sugars, sweets, and also cheese, fats, and butter, should be partaken of sparingly. Alcoholic beverages must be forbidden. Tea and coffee may be allowed only sparingly. Smokers had better give up the use of tobacco. Exercise, outdoor life, sea-bathing, change of climate, etc., are eminently beneficial, and long sea voyages are especially of value. The various mineral springs, especially Carlsbad, are particularly to be recommended. The medicinal treatment should consist of laxative doses of calomel from time to time, alternating with some saline, such as the phosphate of sodium. The preparations of lithia have been generally advised. Piperazin, in 5-grain doses three times daily, is effective in many cases.

OBESITY.

Definition.—This condition is due to an increase in the fats of the body.

Synonyms.—Polysarcia; lipomatosis universalis; corpulency.

Etiology.—The disease occurs in either sex; it is, however, more common in women. It frequently devolops after the menopause. It may be either hereditary or acquired. In the acquired form it results from lack of exercise and from excessive eating, and is especially common in the uric acid diathesis. It results from the use of alcoholic beverages, particularly the malt liquors. It occurs in indolent, phlegmatic individuals, while those of the nervous temperament are not inclined to stoutness. Certain diseases, such as diabetes mellitus and gout, favor the development of fat. It is also common in chlorotic girls and in some of the severe forms of anemia. Corpulency may follow some diseases, such as pneumonia, enteric fever, and neurasthenia, and it also develops after ovariotomies and castration. Deficient oxidation may also be included among the causes. Certain occupations which tend to lessen the normal activities predispose to obesity. The ingestion of large amounts of fluids is liable to cause corpulency.

Symptoms.—The normal activities of the individual are much impaired from the accumulation of fat around some of the organs; their functions become lessened, the circulation is likely to be poor, and degenerative changes may follow in the heart muscle. In some individuals plethora instead of anemia is found. The appetite is perverted, and, as a rule, poor; indigestion and constipation are frequently present. In some instances there is some degree of mental impairment, the individuals complaining of being tired and sleepy. The temperature is usually subnormal. The condition may remain stationary for years.

Prognosis.—Under appropriate treatment the body-weight is sometimes materially reduced, with decided benefit to the patient.

Treatment.—The treatment is in the main dietetic. Food should be partaken of sparingly, and the starches, sugars, and fats must be reduced to a minimum. Systematic exercise and hydrotherapy are useful. Alcoholic beverages, especially

the heavy wines and malt liquors, are to be avoided. Some of the special cures, such as advised at Kissingen and Vichy, are beneficial.

The administration of thyroid extract is attended with some danger, and it should always be cautiously employed. Good results have, however, followed its use.

OSTEOMALACIA.

Definition.—An affection characterized by softening and bending of the bones owing to the solution of lime-salts.

Synonyms.—Mollities ossium; malacosteon.

Etiology.—The cause of the disease is not known. It occurs most often in females, and most frequently between the ages of twenty-five and forty-five. It is very commonly associated with pregnancy. Repeated pregnancies seem to aggravate the affection.

Pathology.—Softening of the pelvic bones occurs early, and produces marked deformities of these bones, but the disease may begin in the bodies of the vertebræ. The acetabulum is forced inward and the iliac bones are flared outward (winged). The lumbar vertebræ are pushed forward and downward, and the sacrum and pubes are also pushed forward. The vertebræ and the femur may undergo extensive softening. Fractures are common in this disease. Absorption of the lime-salts begins at the edges of the trabeculæ and Haversian canals.

Symptoms.—General feebleness and debility, accompanied by pains in the region of the pelvis and in the lower extremities, with a peculiar unsteadiness of the gait, are early symptoms. Kyphosis, lordosis, and scoliosis are common. Parturition becomes difficult. The urine contains an increased amount of lime-salts. The disease is chronic, lasting for many years.

Treatment.—The treatment consists in good hygiene and the use of tonics.

PULMONARY HYPERTROPHIC OSTEOARTHROPATHY.

Definition.—This affection is characterized by deformity and enlargement of the bones, affecting principally those of the hands, wrists, ankles, and feet in persons affected by chronic pulmonary disease.

Etiology.—The disease is rare, having first been described by Marie. It has been noted as occurring in connection with empyema, pulmonary tuberculosis, new growths of the lung, and some forms of chronic bronchitis.

Symptoms.—The principal enlargement occurs in the fingers, which increase both in length and thickness (club deformity), the nails becoming considerably curved and fibrous, often resembling the talons of a bird. The wrists are swollen, and the extremities of the radius and of the ulna are enlarged. The changes in the foot are similar to those described in the hand. The deformity of the bones is not symmetric. There is often swelling of the articular ends of the long bones, and occasionally effusions take place into the joints. Spinal curvature, especially in the dorsolumbar region, is frequent. Much of the pathology is still unknown.

Treatment.—No methods are known which will remedy the affection.

OSTEITIS DEFORMANS.

Definition.—A disease characterized by marked deformity of different bones of the body and by constitutional disturbances.

Etiology.—The etiology of this disease is still unknown. The disease was first described by Sir James Paget, in 1877. It chiefly affects the aged, and is more common in males than in females.

Symptoms.—The deformities may involve many bones, or may be restricted to one, such as the tibia or the femur. This is particularly true early in the disease; later, the vertebræ, the skull, and other bones of the body become involved. Bowing of the long bones and curvature of the spine develop. In some parts of the bone marked absorption is going on, and is slight in other parts. New bone is also deposited in certain areas, therefore a marked deformity results. The disease may persist for a number of years, but it rarely causes death. After the disease has persisted for a while, carcinoma and sarcoma are apt to follow in some parts of the body.

Prognosis.—The prognosis is unfavorable, and no remedy influences the course of the disease.

PART VII.

DISEASES OF THE BLOOD AND OF THE DUCTLESS GLANDS.

ANEMIA.

By the term *anemia* is meant a deficiency of the blood either in its bulk or in certain of its constituents; by *oligemia* is usually meant a deficiency of the blood as a whole; by *oligocythemia*, a decrease in the number of erythrocytes; by *oligochromemia*, a reduction in the hemoglobin.

The classification of anemia is by no means satisfactory; the one which must still be adhered to is that of primary and secondary anemia. By the term **primary**—sometimes called essential, idiopathic, or cryptogenetic—is meant *a disturbance of the blood or of the blood-making organs* (spleen, bone-marrow, and lymphatic glands), *so that the anemia seems the distinctive feature of the disease, while other symptoms appear mainly dependent upon this change.* Under this heading are properly considered chlorosis and pernicious anemia. **Secondary anemia** is due *to some disease acting upon the blood or blood-making organs, the anemia not being the prime feature, but a symptomatic manifestation.* Perhaps a more lucid and scientific classification will be adopted when our knowledge relating to the subject becomes more definite; for example, such a classification as: (1) anemia due to deficient blood formation; (2) anemia due to excessive blood destruction (hemolysis); (3) anemia due partly to both these causes.

SECONDARY ANEMIA.

Synonym.—Symptomatic anemia.

Etiology.—Hemorrhage.—The severity of the anemia from this cause necessarily varies greatly, depending upon whether

the loss of blood be rapid or gradual, and upon the amount. Indeed, some of the severest forms of anemia are encountered from this cause. It can not be stated just how great a loss of blood will produce a fatal issue, as individual predisposition and the rapidity of hemorrhage are factors which determine this. Hemorrhage may occur from many causes. Rapid bleeding is often due to tuberculosis of the lungs, uterine disease, gastric ulcer, rupture of aneurysms, external injury, scurvy, purpura, hemophilia, etc. Slow hemorrhage, which often causes intense anemia, is due to such causes as hemorrhoids, carcinoma, and uterine disease.

Improper Food or Deficiency of Food.—Anemia from this cause may be due to lack of food, to improper food, or to either functional or organic derangement of some part of the gastro-intestinal tract. Such conditions as stricture of the esophagus, due either to cicatrices or to tumors, carcinoma of the stomach, the various forms of gastritis and dyspepsia, may give rise to secondary anemia from failure to properly assimilate food.

Organic diseases, particularly those diseases which cause a constant, long-continued drain of the albuminous materials of the blood, give rise to pronounced anemias. This is so of chronic parenchymatous nephritis, long-continued suppuration, diarrhea, leukorrhea, etc. Malignant tumors also produce pronounced anemia.

The blood, an agent which acts upon tissues through the medium of other tissues, performs one of the chief functions in the complex system of animal life ; therefore a disease of an organ will affect other tissues through the agency of the blood. Diseases of the brain, spinal cord, heart, liver, kidney, lung, pancreas, bone-marrow, spleen, thyroid gland, and lymph-glands, all tend to produce anemia.

Infectious Diseases.—In the acute infectious diseases the blood suffers in a marked degree, principally through excessive hemolytic action, either directly by the action of the specific germ or its toxin. This is particularly true of such diseases as acute rheumatic fever, pyemia, septicemia, diphtheria, pneumonia, enteric fever, etc. The chronic infectious diseases produce a pronounced form of anemia. This is encountered in syphilis, tuberculosis, and other diseases.

Toxic Causes.—Poisons introduced from without or developed within the body produce anemia. Such anemia may be due to lead, mercury, arsenic, copper, etc. Various poisons

may be absorbed through faulty digestion. In gout and rachitis anemia is developed.

Animal Parasites.—These either directly or indirectly produce anemia. The malarial parasite is an example of direct action upon the red blood-cell. The anchylostoma duodenale produces an extreme form of anemia, which is sometimes spoken of as "Egyptian chlorosis." The bothriocephalus latus also gives rise to profound anemia. Other parasites which give rise to anemia are the ascaris lumbricoides, the filaria sanguinis hominis, the oxyuris vermicularis, the cestodes, the distoma hæmatobium and the distoma pulmonalis, the amœba coli, etc.

Pathology.—The state of the blood depends upon the severity and duration of the cause acting and upon the power of blood regeneration, so therefore symptomatic anemia varies from the slightest impairment to the gravest form of anemia, not unlike progressive pernicious anemia. The composite picture of the blood in secondary anemia is by no means easy of description. The anemia depends largely upon the power the individual possesses to regenerate blood.

The fluid and albuminous principles of the blood seem to be quickly restored, the corpuscular elements next, and, lastly, the hemoglobin is replaced. The latter often requires weeks, or even months, before the normal standard is reached. It is for this reason that the greater number of secondary anemias show a more decided decrease in the percentage of hemoglobin than in the number of erythrocytes; hence a low color-index. Indeed, this is sometimes so striking that the anemia is called by some "chlorotic anemia." The latter condition is encountered in tuberculosis, syphilis, and cancer. In some diseases the plasma of the blood is withdrawn in excess of the corpuscular element, so that the erythrocytes are relatively increased, owing to concentration; this is encountered in cholera and in severe diarrheas.

The leukocytes are usually increased in secondary anemia, the most important exceptions being enteric fever, tuberculosis, malaria, measles, and influenza. (See Leukocytosis.) The polymorphonuclear neutrophiles are generally increased both relatively and absolutely; but there are exceptions, for the eosinophiles or the lymphocytes may be the only ones which show the increase. A few myelocytes may appear in the blood, particularly in grave forms of symptomatic anemia, or when the bone-marrow is disturbed from the pressure of tumors, etc.

The erythrocytes in slight anemia show little or no structural

change, but as the severity increases, various abnormalities are encountered. One of the most important changes is perhaps variation in size; macrocytes and microcytes are noticed; the greater number of cells seem to be under the normal size. Poikilocytosis also manifests itself. Nucleated cells may be found, particularly normoblasts. In the regeneration of blood after copious hemorrhage great numbers of normoblasts appear in the circulation, which, however, soon disappear as the blood tends to reach the normal. Degenerated cells are sometimes found.

The gross appearance of the blood presents wide variations; it is commonly paler and more fluid; the specific gravity is generally reduced, the watery elements being increased. The alkalinity is never markedly changed, although it is usually somewhat diminished.

Symptoms.—Symptoms depending upon secondary blood changes are not always in evidence and at other times pronounced. It is evident as the blood becomes impaired that either it fails properly to absorb or to carry to the tissues nourishment, oxygen, or other materials necessary for metabolism, or it fails to deliver to the excretory organs the waste products; definite symptoms therefore arise from the anemia. The symptoms produced by the anemia may, however, be masked by the disease in question.

Pallor of the skin is one of the signs of symptomatic anemia; but this is very deceptive at times. *Pallor of the mucous membranes* (the conjunctivæ and lips) is a more definite indication. In rare cases even the pallor of the mucous membranes is not a definite sign; an instance of this is leukocythemia. Pallor depends rather upon reduction of hemoglobin than of erythrocytes.

Shortness of breathing and palpitation of the heart are constant and important symptoms. The dyspnea upon exertion is due to the deficiency of hemoglobin, and, in the severer forms of anemia, also due to a diseased heart muscle. Palpitation, like shortness of breath, develops upon the slightest exertion or the least excitement. Headache, throbbing in the head, tinnitus aurium, dizziness, and fainting are often seen, and are due to the impoverished blood or insufficient blood supply to the brain. Restlessness, peevishness, and irritability of temper are common, even delirium, coma, and convulsions may occur. Neuralgia is also a symptom, and is due to the poverty of the blood.

In pronounced, long-continued anemia the heart muscle tends to become fatty and the chambers dilate, giving rise to distinct physical signs. The apex-beat may become displaced to the left, and may be more diffuse, and, on account of palpitation, it is more forcible, but most often the impulse is weaker than normal. With dilatation, relative incompetency of the mitral and tricuspid valves may develop. Upon auscultation, murmurs may be heard in any area, the one most commonly noticed being a soft, systolic, blowing murmur in the pulmonary area. A venous hum (*bruit de diable*, or nun's murmur) is frequently heard over the veins of the neck.

Edema, chiefly of the feet, and sometimes of the face and hands, is seen only in the profound forms of anemia, and is probably to be ascribed to changes in the composition of the blood and to cardiac disturbance. If the blood-vessel walls undergo fatty change, they are liable to rupture, hemorrhage following. This is observed only in extreme forms of secondary anemia, more commonly in primary anemia (progressive pernicious anemia). Hemorrhage may occur in the skin, subcutaneous tissues, and retina, into the serous cavities, and from the mucous membranes.

Digestive symptoms are also present: dyspepsia is common; the appetite is impaired; the tongue is coated; the bowels are most often constipated, although diarrhea may exist. Loss of body-weight is not a symptom of anemia, but rather of the underlying disease. *It may be said that when there is very little or no loss of weight, the anemia in question is of a primary nature, while if the nutrition of the body suffers greatly, and there is marked emaciation, the anemia is probably secondary.* Weakness is pronounced; febrile attacks, known as "anemic fever" or "essential fever of anemia," are common. The temperature range may be intermittent, remittent, or subcontinuous. The urine is commonly paler than normal, although much depends upon the underlying cause. Menstrual disturbances, especially amenorrhea, are frequent, but menorrhagia may be present, and in some cases may be the cause of the anemia. The symptoms and signs of rapid profuse bleeding are the same as those of shock.

Prognosis.—This depends upon the underlying cause.

Treatment.—The treatment should be directed to the cause. If due to hemorrhage, measures speedily to control this should be instituted. If due to improper or deficient food, this should be corrected. In organic disease the underlying

condition must first be treated; there is often little hope for cure in these cases. In the infectious diseases the anemia is, as a rule, treated in convalescence. When caused by toxic substances and animal parasites, these should be, if possible, eliminated. The drugs which have proved most efficient are iron, arsenic, cod-liver oil, bone-marrow, etc.

CHLOROSIS.

Definition.—Chlorosis is a form of primary anemia, affecting chiefly the female sex at the time of puberty or in early womanhood, and characterized by marked oligochromemia.

Synonyms.—Chloremia; chloranemia; green sickness; morbus virgineus.

Etiology.—This disease almost exclusively affects females, rarely, if ever, males. It develops at about the time of puberty; occasionally later in life, when it is called "chlorosis tarda." The disease is known in all races and every climate. Certain occupations predispose to the disease. It is common in those closely housed, such as mill girls, school children, and those employed in factories or stores. Sedentary habits, lack of exercise, fresh air, and sunlight, mental anxiety, change of climate, and homesickness also predispose. The city seems to be more favorable for its development than the country. Heredity seems to play some part in the cause. It is common in girls who emigrate. There is a relationship between tuberculosis and chlorosis, as girls who have a scrofulous tendency often become chlorotic. The disease is said by some to be more common in blonds, but this is doubtful. There are many theories as to the cause. Virchow observed hypoplasia of the arterial system, especially narrowing of the aorta. Sir Andrew Clark supposed that it was due to auto-intoxication from constipation. Ulceration of the stomach has also been suggested as a cause. Pick believed that it was due to the absorption of some poisonous substance from a dilated stomach. Lloyd Jones regards chlorosis as an exaggeration of some physiologic condition which occurs in the blood of healthy females at the time of puberty, and which shows itself in many women at each menstrual period. He also observed that it occurred in large families.

Pathology.—The heart and the arterial system may show hypoplasia; the genital organs may be imperfectly developed.

The blood, however, shows the most marked pathologic changes.

Pathology of the Blood.—The gross appearance of the blood is altered, it being remarkably fluid, flowing freely, and it is of a pale red color. There is a tendency to coagulation in the blood-vessels, but extravascular coagulation is retarded. The alkalinity is slightly increased. The specific gravity of the blood is reduced, but the specific gravity of the serum is sometimes higher than the normal; the number of erythrocytes is normal or slightly decreased; the hemoglobin shows a constant marked reduction; the color-index is constantly low. The leukocytes are normal as to number; however, sometimes the lymphocytes are increased at the expense of the polynuclear elements. The red blood-cells are undersized, and in extreme cases may show variation in size and outline. Nucleated cells are occasionally found, being usually of the normoblastic type. A few myelocytes may rarely be present. The blood plates are usually increased.

Symptoms.—The disease is gradual in its onset, the patient losing color, complaining of dyspnea, weakness, palpitation of the heart, and constipation. In a few instances the color of the lips and cheeks is not lost, but is quite high, the term *chlorosis florida* being applied to these cases. The appetite becomes perverted; there is usually a craving for sour food, such as pickles, or for sweets; often school-girls are found to be eating chalk. Dyspeptic symptoms are common. As the disease progresses the pallor becomes extreme. The skin is of a yellow-green color; hence the term "green sickness," or "chlorosis." The green hue is more apparent in dark-complexioned than in fair individuals. Extreme weakness and giddiness accompany the pallor. In chlorosis it is sometimes noticed that there is an absence of horizontal folds in the forehead when the patient is suddenly asked to look up without raising the head; this is known as "Joffroy's sign." Slight puffiness or edema of the face, hands, and ankles may appear; the conjunctivæ are pale and the sclerotic coat of the eye becomes bluish in color. Menstrual disturbances, especially amenorrhea, are common; occasionally there may be slight pyrexia. Loss of flesh is not apparent; in some cases a gain in weight will occur. The respiration is quickened; a rapid heart action will be noticed.

These symptoms are probably due to the defect of coloring-matter in the blood, nature making an effort to carry the

hemoglobin rapidly so as to make up for the deficiency in the amount, hence giving rise to palpitation, dyspnea, headache, neuralgia, giddiness, slight edema, etc. The fats are probably not oxidized, therefore the weight does not decrease. Examination of the heart will most often reveal a soft, systolic murmur, heard loudest at the pulmonary area. A systolic murmur is sometimes heard over the subclavian artery. Over the veins of the neck a continuous murmur is heard, called the venous hum, *bruit de diable*, or nun's murmur.

Diagnosis.—The occurrence of the disease in young females with digestive, menstrual, and vascular derangements, and the blood examination, are necessary to diagnosticate the condition.

Secondary Anemia.—This condition often shows the same blood changes, and it is sometimes called chlorotic anemia, but the leukocytes are usually increased, less commonly normal, and rarely decreased. The clinical history is necessary for diagnosis.

Prognosis.—This is always favorable unless complications develop.

Treatment.—Rest in bed is important, often bringing about a complete recovery without the administration of drugs. Fresh air and sunlight are beneficial, and food should be nutritious. Iron is indicated and is of great use in the treatment. Arsenic, bone-marrow, hydrotherapy, and oxygen inhalations are sometimes recommended. The patient usually recovers in from three to six weeks.

PERNICIOUS ANEMIA.

Definition.—This is a primary anemia, characterized by a marked decrease in the number of red blood-cells, by fatty degeneration of the heart, liver, and kidneys, and by a peculiar lemon-yellow discoloration of the skin.

Synonyms. — Progressive pernicious anemia; idiopathic anemia; essential anemia; corpuscular anemia; myelogenic anemia.

Etiology.—Addison, in 1855, described the disease, which is sometimes called "Addison's anemia." Most frequently this disease affects males. It is most common in middle life. The cause has not been determined. Pregnancy and parturition are predisposing causes. Atrophy of the gastric tubules has been regarded as an etiologic factor. Banti suggested that it was

due to a lesion of the sympathetic nerves, and termed it "ganglionic anemia." The presence of intestinal parasites, such as anchylostoma duodenale and the bothriocephalus latus, often produces extreme anemia. Occupation and habit do not seem to play an important part among the predisposing causes.

Pathology.—The skin shows a characteristic lemon-yellow discoloration. The fats are well preserved and of a light yellow color. The spleen may be somewhat enlarged and may show pigmentation. The heart, liver, and kidneys reveal fatty degeneration and iron pigmentation. The stomach is often small and the gastric tubules are atrophied. Hemorrhages may be present in the retina and in other parts of the body, such as the skin and the gastro-intestinal and respiratory tracts. The bone-marrow almost constantly shows changes, becoming softened, reddened, or lymphoid in character. Posterior sclerosis of the spinal cord may be present.

The blood shows marked changes; it is watery and pale red, sometimes resembling weak coffee. It is often difficult to procure, as the tissues are almost bloodless. The specific gravity is decreased and the coagulation retarded; the erythrocytes show a marked reduction in their number—in extreme cases 500,000, or a count as low as 143,000 to the cubic millimeter has been recorded. Commonly, 1,000,000 to the cubic millimeter are found. The hemoglobin is also greatly reduced, but the percentage being relatively higher than the colored corpuscles, the color-index, therefore, is above the normal. The leukocytes are normal or reduced in number.

Lymphocytosis is quite constant, a few myelocytes usually being present. The red blood-cells vary greatly in size, the majority of them being somewhat larger than normal, and poikilocytosis is marked—more constant than in any other disease. Nucleated red cells are found, mostly of the megaloblastic type. Polychromatophilic changes, and occasionally shadow corpuscles, are noticed.

Symptoms.—The disease is very gradual in its onset. The patient first notices the extreme pallor, or is informed of it by friends. Shortness of breath, weakness, palpitation of the heart, giddiness, and headache are early symptoms. After some time the weakness becomes extreme, and the patient must discontinue work and consult his physician. Often the disease is ushered in by gastro-intestinal disturbances, as profuse diarrhea and vomiting. Loss of weight is not marked or does not occur. A sense of discomfort, which may amount

to pain, is sometimes experienced in the chest. Hemorrhages may occur in the retina, producing disturbances of vision. Epistaxis, hemoptysis, or hematemesis is present, especially late in the disease. Purpuric symptoms and edema of the face, hands, and feet may arise.

On examination of the patient the skin shows the characteristic lemon-yellow discolorations. The heart-sounds are weak and hemic murmurs are common. The pulse is weak and often irregular. The spleen may be slightly enlarged. The symptoms of the disease gradually become more grave, and death results; or, after prolonged treatment, apparent recovery may seem to ensue, only to be followed by another relapse, when the patient succumbs. Two or three relapses may occur. In the course of pernicious anemia fever often develops (termed "anemic fever"). Constipation, less commonly diarrhea, is present.

Diagnosis.—The diagnosis is by no means easy, and must be made from the history and the blood examination. The disease is often mistaken for obscure malignant growths, renal disease, and other grave affections. The examination of the urine will reveal true kidney lesions.

Prognosis.—Always grave; death results in from a few months to a few years; apparent recovery, followed by relapse, is common.

Treatment.—Rest in bed is essential, and easily digested, nutritious food should be given. Medicinally, arsenic has been found to be of most value, given in the form of Fowler's solution, in ascending doses. Patients often bear large amounts. If this drug is not well borne, iron may be substituted; the latter, however, has not proved of much service in the treatment. Bone-marrow is also useful. Stimulants, such as strychnin and alcohol, and inhalations of oxygen are of advantage.

LEUKEMIA.

Definition.—Leukemia is a disease in which the white blood-corpuscles are greatly increased in number; the percentages of the various forms also differ widely from the normal. It is characterized anatomically by changes in the spleen, lymphatic glands, or bone-marrow, singly or combined.

Synonym.—Leukocythemia.

Varieties.—Splenomedullary (or lienomedullary) and lymphatic.

Historic Notes.—The disease was first described in 1845, by Bennett, as a suppuration of the blood, and very shortly afterward Virchow published a case, the condition being observed independently. The latter also pointed out that the numerous white corpuscles found in the blood were leukocytes, and termed the condition leukemia.

Etiology.—All races are susceptible, but, according to Eichhorst, it is more likely to affect the Jews. It is also more frequent in low life than in the upper ranks of society. The disease has been found in all parts of the world; it is, however, very rare.

The splenomedullary variety is more frequent in adults, while lymphatic leukemia is said to be more prevalent in children. Heredity seems to play some part in the causation. Pregnancy and parturition are also factors. According to Gowers, 20% of the cases of leukemia are preceded by malaria. Syphilis and injury are perhaps predisposing. The exciting cause has not been determined. It has been suggested that it is of an infectious nature. Löwit claims to have discovered an ameba in the blood. This is by no means settled.[1]

Pathology.—In the splenomedullary variety the spleen is found to be tremendously enlarged, weighing ten pounds or more. The capsule is thickened and the surface of the organ is somewhat irregular. On section, it is very firm, especially in advanced cases. The color of the pulp is reddish-brown. Infarcts are common.

This enlargement is due to the proliferative changes of the leukocytes, lymphoid tissue, and usually the connective tissue. Charcot-Leyden crystals may be found in the organ. The bone-marrow changes are the following: The bone-marrow of the spongy and long bones becomes altered early in the disease—softer, and later almost semiliquid (lymphoid or pyoid); the fats are also replaced by the proliferation of cells. These changes are due to the excessive multiplication of nucleated red blood-corpuscles (showing the various stages of mitosis), marrow-cells, and giant cells. There are also many lymphoid corpuscles. The blood changes are probably largely dependent upon the marrow disturbance. The liver may be enlarged and may contain collections of leukocytes in various parts; the kidneys may also contain these nodules.

[1] "Centralblatt für Innere Medicin," No. 19, 1900.

Hemorrhages are common in various organs and from the skin and mucous surfaces in both types.

In the lymphatic variety groups of lymph-glands (but not necessarily all chains) are enlarged, owing to hyperplasia of the lymphoid cells, and the spleen is slightly increased in size.

Pathology of the Blood.—In the splenomedullary variety the gross appearance is altered, occasionally being milky in character; the specific gravity is decreased, the alkalinity is somewhat diminished, and coagulation is slightly retarded. The erythrocytes usually show a slight reduction in number, occasionally being normal; and in some cases, especially those of long standing, a marked decrease is present. The hemoglobin is diminished somewhat more than the percentage of red cells. The color-index is usually below normal. The leukocytes show an enormous increase in the ordinary case—from 250,000 to 400,000 or more. In extreme cases the colorless blood-cells almost equal the number of erythrocytes. The estimation of hemoglobin by the color test is sometimes difficult, on account of the milky appearance of the blood (due to the increased number of leukocytes). An examination of the stained films will reveal large numbers of myelocytes, composing from 30% to 50% of all the leukocytes. Finely granular basophilic cells are met with, and some have observed mast-cells (coarsely granular basophilic cells). Nucleated red blood-cells, usually of the normoblastic type, are more frequent in this variety of leukemia than in any other known condition, and poikilocytosis and degenerative changes may also be found.

In the lymphatic variety the gross appearance of the blood may show very slight change, or may resemble the variety just described. Oligocythemia and oligochromemia are more pronounced than in the splenomedullary form. The leukocytes show a marked increase, from 50,000 to 200,000, but not to the extent found in the splenomedullary type. Examination of the stained films reveals an enormous increase in the lymphocytes; in some cases the larger forms predominate, in others the smaller. They usually constitute the greater proportion of the leukocytes, sometimes as high as from 97% to 98%. A very few myelocytes and nucleated red blood-cells are rarely met with.

Symptoms.—The onset of this disease is insidious, although acute varieties are found which last from a week to three months, ending fatally.

The early symptoms are weakness and loss of weight. In some cases there is marked anemia, but in others this symptom is completely absent.

In the splenomedullary variety fullness and a sense of discomfort in the abdomen will be noticed. Gastro-intestinal disturbances may arise, probably from pressure. Patients have consulted physicians for dyspeptic complaints when suffering from leukemia.

In the lymphatic type the glands become enlarged, affecting chiefly those of the neck, the axilla, and the inguinal region. This enlargement is pronounced or slight, and the spleen is but little increased in size. Shortness of breath will develop as the disease progresses; edema of the feet, ankles, face, and hands will appear in advanced stages; irregular fever and dimness of vision may arise.

In the splenomedullary variety tenderness over the bones—such as the long bones of the extremities, the sternum, and the ribs—develops in many cases. On examining the patient, in the splenomedullary variety, the spleen is found to be greatly enlarged, distending the abdomen, widening the base of the chest, and, indeed, in some cases apparently filling the entire abdominal cavity, so that it reaches to the right anterior superior spine of the ilium. The heart is displaced upward. The liver in many cases is enlarged, the lower border of liver dullness extending far beneath the costal margin. The urine is commonly slightly albuminous, and may contain blood. The general appearance of the patient usually indicates anemia, but there are cases apparently quite robust, termed "leukemia plethora." In the lymphatic variety the anemia is always more pronounced. Hemic murmurs may be present in both these varieties, but are more likely to occur in the lymphatic forms. Purpuric manifestations and hemorrhages may also occur.

Splenomedullary leukemia, which is the most common type, usually runs a course of from one to three years. Treatment may have a distinct beneficial effect, the patient gaining strength, the spleen diminishing in size, and the leukocytes decreasing in numbers, in some cases reaching the normal. The condition invariably returns, and death results usually after a number of relapses.

Diagnosis.—The direct diagnosis depends upon the enlargement of the spleen or lymphatic glands and upon the characteristic blood changes. Indeed, in the splenomedullary variety

the blood picture is typical; the great number of leukocytes—a large percentage of these being myelocytes—and many nucleated red blood-cells revealing the condition. In the lymphatic form an increase in the lymphocytes and an almost entire absence of myelocytes and nucleated red blood-cells are conspicuous.

Hodgkin's disease may be differentiated from leukemia by an absence of the enormous increase in the number of leukocytes and by the slight, if any, enlargement of the spleen. Splenic tumors are easily differentiated by an examination of the blood.

Prognosis.—This is very unfavorable, death generally resulting in from one to three years in the chronic form and in from a few weeks to three months in the acute.

Treatment.—Rest in bed is desirable and a nutritious diet should be given. Arsenic should be administered early, preferably in the form of Fowler's solution, the dose being increased to the point of tolerance; this drug seems to have a marked beneficial effect. The treatment must be continued in order to hold the disease in check, but after a time, in spite of treatment, the condition of the patient will grow worse. The discontinuance of treatment will cause a return of the symptoms. It should always be remembered that arsenical pigmentation of the skin develops after prolonged administration of this drug. Ergot has been recommended; bone-marrow and iron are also found to be useful, especially when arsenic is not well borne. Oxygen inhalations may be beneficial.

HODGKIN'S DISEASE.

Definition.—This is a progressive disease, characterized by hyperplasia of the lymphatic glands, at first local, then becoming general, accompanied by loss of weight, weakness, and anemia.

Synonyms.—Pseudoleukemia; lymphadenoma; adénie; malignant lymphoma; lymphatic anemia.

Etiology.—The disease was described by Hodgkin in 1832. This is a rare disease. It occurs most frequently before the age of forty; males are more susceptible than females in the ratio of 3 to 1. Mental anxiety, ill health, and poor nutriment seem to play some part as predisposing causes. Malaria, rickets, and syphilis also appear to predispose. The exciting cause has not been determined; it is, no doubt, some irritant which causes the proliferation of the lymphoid elements.

Pathology.—The enlargement of the lymphatics is at first local, and often superficial, affecting most frequently the anterior or posterior cervical chains on one side. The axillary glands are sometimes first enlarged, and more rarely the inguinal. The intrathoracic or abdominal gland may sometimes be the primary seat. The condition remains local for some time, then, becoming general, the remaining lymphatic glands become enlarged, being those of the mediastinum, posterior peritoneal and mesenteric glands, etc. Early the glands are slightly enlarged, are freely movable, and are separated from one another; later they greatly increase, often reaching the size of a cocoanut; they may become adherent to one another.

As the disease progresses and lymphoid deposits begin to appear in many organs, the spleen, being most commonly affected, is enlarged as a result. The glands may be either hard or soft; they rarely, if ever, caseate or ulcerate, and when this does occur, it is limited and very slight, and had better be considered an associated or secondary manifestation. The bone-marrow usually becomes lymphoid in character.

Microscopic examination of the glands reveals proliferation of the lymphoid elements; the interstitial substance is marked in some cases, causing a hard condition of the glands, while this is almost entirely absent in others.

According to Bramwell, "it is important to note that, although in typical cases of Hodgkin's disease the glandular enlargements present the pathologic characters which have just been described, in some cases in which the glandular enlargements during life present all the characteristic *clinical* features of Hodgkin's disease, the enlarged glands are found after death to be caseous and tubercular." He further adds: "I have seen several cases of this kind, and I have been so impressed with the difficulty there is in some cases, more especially in children and in young subjects, of differentiating during life the glandular enlargement due to Hodgkin's disease from that due to tubercle, that I now have great hesitation in committing myself to a definite diagnosis of Hodgkin's disease and in excluding tubercle, unless the spleen is distinctly enlarged, or unless there is evidence of the presence of lymphoid deposits in other organs and tissues."

The Blood.—The anemia in Hodgkin's disease is commonly of the chlorotic type, mild in the beginning and severe toward the end of the disease. Poikilocytosis and nucleation of the red

blood-cells may occur in extreme degrees of anemia. The leukocytes are usually normal; they may be slightly increased or decreased. It is common to see a slight increase in the percentage of the polynuclear elements, rarely in the lymphocytes. It is claimed by some that pseudoleukemia terminates in leukocythemia.

Symptoms.—The onset is insidious, and usually pursues a long-continued course, rarely an acute one. Weakness begins to show itself, the glandular enlargement appears, emaciation soon follows, and anemia also develops. Palpitation of the heart may arise as a result of the anemia. There is loss of appetite, arising from the impaired nutrition. Very soon in the course of the disease fever, irregular or hectic in type, develops. The glandular enlargement most frequently affects the cervical chain, either the anterior or the posterior, unilateral or bilateral. These glands are freely movable and painless, and present no signs of acute inflammation. They increase in size, becoming somewhat fused together, and rarely soften and suppurate. This enlargement may be local for some time,—indeed, for a year or more,—then it spreads to the axillary, inguinal, retroperitoneal, bronchial, mediastinal, and mesenteric glands. The enlargement in many instances is extreme, so that it becomes impossible for the patient to place the arm alongside the body or to draw the head down upon the chest. In 75% of the cases, according to Gowers, the spleen will become slightly enlarged, so that it might be readily palpated. Pressure symptoms might arise at any time from the glandular enlargement. Toward the end of the disease profound anemia and cachexia appear. Weakness and emaciation are marked and hemic murmurs are to be heard over the heart. Loss of appetite and dyspeptic symptoms usually indicate lymphoid deposits in some part of the gastro-intestinal tract.

The course of the disease is, as a rule, chronic, lasting from one to three years, death almost invariably occurring, either as a result of the gradual failure of the vital powers or from pressure symptoms upon the bronchus, the trachea, the larynx, or the esophagus. Cases may terminate from intercurrent complications.

Diagnosis.—The diagnosis of Hodgkin's disease is difficult in the early stages, but less so when the disease is fully developed. The general glandular enlargement, commonly accompanied by increase in the size of the spleen and by

severe anemia, is characteristic. A blood examination is necessary to differentiate it from leukocythemia.

Differential Diagnosis.—Hodgkin's disease is often with difficulty differentiated from tubercular adenitis. Tuberculosis often affects the glands of the neck, especially those of the submaxillary chain, most frequently unilateral, and showing a distinct tendency to softening and breaking down. Early life predisposes to, and tuberculosis of the lungs often accompanies, the condition. The glandular enlargement persists for a long time and shows no tendency to become general, while in Hodgkin's disease the reverse is true. The enlarged glands may, however, fuse together, somewhat resembling tubercular adenitis, but do not show a tendency to break down or to caseate. The blood will not show distinct points of differentiation between these two conditions.

The differential diagnosis of Hodgkin's disease from sarcoma of the lymphatic glands is often impossible, especially in the early stages. Sarcoma spreads by means of the blood currents, thus causing the growth to arise in almost any part of the body, very often quite remote from the original seat, while in Hodgkin's disease the enlargement spreads and seems to follow the course of the lymphatic glands more or less closely. The blood may at times show points of differentiation—a leukocytosis of from 20,000 to 50,000 is suggestive of sarcoma; but if this does not exist, it is quite impossible to show any point of importance from the blood examination. Again, the spleen is usually enlarged in Hodgkin's disease, while it is not in sarcoma.

Carcinomata of the lymphatic glands may be differentiated from Hodgkin's disease by the following: The glands are always secondarily involved, and the primary seat of the new growth will give the characteristic symptoms, depending upon the locality. The splenic enlargement does not usually exist, and the disease is most frequent after middle life.

The differentiation of Hodgkin's disease from lymphatic and splenomedullary leukemia is made by examination of the blood.

Prognosis.—The prognosis is grave.

Treatment.—If the condition is diagnosticated early, surgical interference may be tried. Medicinal treatment is of very little avail, arsenic being the only drug which seems to retard the disease somewhat. It should be given in the form of Fowler's solution, well diluted, in ascending doses, until the physiologic point is reached. Tonics, such as cod-liver oil, quinin, and iron, will be found beneficial.

SPLENIC ANEMIA.

Definition.—This is a disease characterized by enlargement of the spleen and by marked anemia of the chlorotic type, without increase in the number of leukocytes, and always terminating fatally.

Synonyms.—Splenic pseudoleukemia; splenic cachexia; lymphadenoma splenicum.

Etiology.—Little is known of the etiology, and some still question the clinical identity of this disease. It was described by Banti, in 1882, and is sometimes known as Banti's disease. The disease is very rare; males are affected more often than females in the proportion of 4 to 1, in adult life between the ages of twenty and fifty.

Pathology.—Emaciation is not marked, the bodily fats being well preserved. The spleen is enlarged, in some instances weighing as much as seven pounds. Its consistence is firm and its color reddish-brown; the splenic capsule is sometimes thickened. Infarcts have been noted in the organ. Upon microscopic examination it is found that the fibrous connective tissue is greatly increased and that many of the Malpighian bodies are replaced by the cicatricial tissues. The splenic pulp is much reduced. In short, the organ is in an atrophic state. The liver is sometimes slightly enlarged, and shows beginning cirrhotic change. The bone-marrow and the lymphatic glands are normal. Hemorrhages are sometimes found in various parts of the body. The blood reveals an extensive reduction in the number of erythrocytes, but always a more marked decrease in the hemoglobin, hence a low color-index (chlorotic type). The leukocytes show no absolute increase, but when complications are present, they may be slightly increased. A relative lymphocytosis has been encountered. The red blood-cells may show nucleated forms and some degree of poikilocytosis.

Symptoms.—The onset is insidious, and is marked by symptoms which are referable to anemia, such as shortness of breath, palpitation of the heart, dizziness, considerable loss of appetite, nausea, vomiting, and constipation, although in some instances diarrhea is present. Hemorrhages from the mucous membranes and into the tissues may be observed. Later, the spleen becomes enlarged, the organ may extend beyond the median line below the level of the umbilicus, and pain in this region is common, being due to *secondary involvement*

of either the pleura or the peritoneum. Irregular fever (anemic fever) usually accompanies the disease. The heart shows some degree of dilatation, and hemic murmurs are usually heard. The course of the disease may be interrupted by a temporary arrest of the symptoms, but very soon a relapse takes place; the entire duration varies from six months to two years. Instances are on record where the disease has lasted as long as four and a half years.

Diagnosis.—This depends upon the enlargement of the spleen and upon the examination of the blood. Pernicious anemia is distinguished from this disease by the absence of great enlargement of the spleen and by the blood examination. In Hodgkin's disease the lymphatic glands are involved. In splenomedullary leukemia the blood examination is sufficient for diagnosis. The enlargement of the spleen due to atrophic cirrhosis of the liver is distinguished from this condition by the other signs of portal congestion (ascites, hemorrhoids, etc.). The enlargement of the spleen in malaria is easily diagnosticated on account of the presence of the plasmodium in the blood.

Prognosis.—The prognosis is hopeless.

Treatment.—The treatment is that of other pronounced anemias, but no remedy is on record which has a decided effect.

ADDISON'S DISEASE.

Definition.—A disease characterized by asthenia, feebleness of the heart action, nausea, vomiting, and bronzing of the skin and sometimes of the mucous membranes, associated with lesion of the suprarenal capsules.

Synonyms.—Morbus Addisonii; melasma suprarenale; bronzed-skin disease.

Etiology.—This disease was described by Dr. Addison, in 1849. It is most common between the ages of twenty and forty; it is rare in early or in advanced periods of life. It is more prevalent in males. Blows and injuries to the back occasionally seem to be exciting causes. The disease is almost constantly associated with tubercular lesions of the suprarenal bodies.

Pathology.—The lesion is usually bilateral, occasionally unilateral. Rarely no lesion of the adrenals will be found. In a large number of the cases both suprarenal bodies are tubercular and enlarged, although occasionally smaller than normal. They are firm and nodulated, irregular in outline,

and show the characteristic caseous necrosis of tuberculosis. Interstitial change, showing a preponderance of fibrous connective tissue, sometimes exists. Simple atrophy and sclerotic and fatty changes of the adrenals have been found in connection with the disease, and occasionally the entire organ may be replaced by fat. These lesions seem to excite inflammatory induration in the sympathetic nerve plexus around the organ, in this way affecting the semilunar ganglion. The latter changes, however, are not constant. Carcinoma and sarcoma of the suprarenal capsules may rarely be associated with Addison's disease.

Some authors favor the nervous theory : that the symptoms are due to disturbances of the abdominal sympathetic nerves ; while many hold the view that the manifestations of the disease are due to insufficient secretion of the suprarenal bodies. Bramwell notes on theoretic grounds that the symptoms of Addison's disease are partly due to the destruction of the capsules and partly to secondary disturbances in the nerves that surround and are in connection with the suprarenal bodies ; and he sees no reason why any lesion of the capsules, provided only that it is sufficiently destructive, sufficiently chronic, and, perhaps, sufficiently irritative in character, may not produce the symptoms of Addison's disease.

Unilateral disease of the suprarenal capsules, tuberculous or otherwise, may not necessarily give rise to the clinical manifestations. When the symptoms are present and only one of the organs is diseased, there is probably some implication of the nerve structure surrounding it. The skin discoloration appears to be an accumulation of the normal pigmentation. The cells of the stratum Malpighii contain the pigment. The spleen is occasionally enlarged. The heart may present marked atrophy.

Symptoms.—The disease is insidious in its onset, and is early characterized by *asthenia* and by *feebleness of the heart's action*, the skin pigmentation generally following these symptoms, but occasionally it is the first sign. The asthenia seems to be the result of the lesion of the suprarenal bodies. As the disease progresses, weakness of both mind and body becomes extreme. Shortness of breath and palpitation of the heart are early symptoms. The heart-sounds and the pulse are weak in nearly all cases. Symptoms of exhaustion and depression may arise, especially on slight exertion. The disease is of a chronic nature. Later the palpitation of the

heart and shortness of breath become extreme, and fainting, disturbances of vision, and cerebral symptoms are liable to arise. Irritability of the stomach, retching, nausea, and vomiting are symptoms of importance, due to the asthenia. The bowels are usually constipated. Pain may be present in the small of the back and in the abdomen.

Pigmentation of the skin and mucous membranes is the most striking and important sign.

This develops gradually, and usually progresses as constitutional symptoms increase. The discoloration is first of a light bronzed color, and becomes deeper as the disease advances. The pigmentation is usually most marked in regions of the body where normal pigmentation is prominent, as the areola of the nipple, around the genital organs, in the axillæ, in the groins, upon the backs of the hands, and around the umbilicus.

Late in its course the entire body may be of a uniform bronzed color, giving the patient the appearance of a mulatto. The mucous membranes, especially those of the gums, lips, and tongue, frequently show pigmentation. In rare instances the pigmentation may be absent, but the other characteristic symptoms are present.

Emaciation is not a marked symptom, and is usually slight. The temperature range, as a rule, is normal or subnormal during its course. The anemia develops gradually, there being a decided decrease in the number of erythrocytes and in the percentage of hemoglobin, or the latter may show a greater reduction than the corpuscles, producing the chlorotic type of anemia. The white blood-cells are, as a rule, normal. The urine may present changes, but they are not characteristic.

The disease terminates fatally, either from asthenia, or, in some instances, the patient sinks into the "typhoid state."

Diagnosis.—The diagnosis, as a rule, is easy. The asthenia, weakness of the heart, vomiting, the presence of anemia without marked emaciation, and the pigmentation of the skin—easily warrant a positive diagnosis. In atypical cases, when pigmentation is slight or absent, or in those cases which are early marked by pigmentation without constitutional symptoms, the diagnosis is difficult or impossible.

Differential Diagnosis.—Various diseases may mislead one's diagnosis, such as vagabond's discoloration of the skin, due to pediculi. Chronic pulmonary tuberculosis may

be associated with pigmentation of the skin. Nitrate of silver discoloration is usually darker, and is associated with the treatment by this drug which has been long administered. Arsenical discoloration may be distinguished by a general mottling, associated with the long-continued administration of arsenic, very frequently seen in cases of leukemia. The pigmentation sometimes seen in malaria, especially in the cachectic form, and in scurvy, pregnancy, syphilis, malignant diseases, and exophthalmic goiter is, as a rule, readily distinguished from that of Addison's disease. Chronic peritonitis and sunburn may also be easily rcognized, as the constitutional symptoms are absent. Pernicious anemia may be differentiated from Addison's disease, as the skin discoloration is lemon-yellow, and also by examination of the blood. Hypertrophic cirrhosis of the liver causes a deep yellow discoloration, but this should not be mistaken for Addison's disease, as the liver is tremendously enlarged and the disease is associated with other symptoms of jaundice.

Prognosis.—The prognosis is grave, death resulting in about two years.

Treatment.—The treatment consists in rest and in avoidance of overexertion and of mental emotions. The diet should be light, nutritious, and should consist principally of milk, white meats, and eggs. Alcoholic stimulants may be of use. Cod-liver oil, iron, and strychnin are also of use. Extract of suprarenal capsule has, in a very few instances, been found valuable in the treatment of this disease.

SCURVY.

Definition.—Scurvy is an affection characterized by anemia, by swollen, tender, and bleeding gums, by manifestations of purpura, and by great prostration, due to improper food.

Synonym.—Scorbutus.

Etiology.—The disease was very common in former times, occurring particularly among sailors during long voyages, the cause being lack of fresh vegetables and of proper food. Since the introduction of steam in navigation the disease has become extremely rare. The exciting cause is undoubtedly faulty diet, the principal errors being an excess of salt meat and fish, the use of stale and tainted food, *and the lack of fresh vegetables, fruits, and so on.* It is probable that the antiscorbutic ele-

ments are the potash and some of the organic acids. All ages are susceptible, not even infancy being exempt. Some conditions predispose to scurvy, such as anemia, inanition, chronic intestinal disease, dysentery, chronic malaria, alcoholism, and syphilis. The disease is rare in this country, but is common in certain parts of Russia. It occurs in times of famine and during long sieges. Scurvy is regarded by some as an infectious disease, but no positive evidence confirms this view.

Pathology.—The most constant lesions are those found in association with the gums, these being softened and ulcerated, with hemorrhages into the tissues, and in severe cases the teeth become loosened and fall out. Hemorrhages are encountered in many mucous membranes also. Ulcers sometimes appear in the ileum and in the colon. The skin, the joints, the kidneys, the serous membranes, or the muscles may be the seat of blood extravasations, and some of the internal organs may reveal granular degeneration. The blood shows no distinctive changes except those of anemia.

Symptoms.—The disease comes on insidiously; rarely is the onset acute. As a rule, there is a history of prolonged prostration, anorexia, and lassitude, the gums finally becoming sore, soft, and spongy, and bleeding readily. The teeth become loosened, and in severe cases drop out. Ulceration of the gums develops and ecchymosis appears. The breath becomes foul and offensive and the tongue may be swollen. There may be free bleeding from some of the mucous membranes, and epistaxis is common. Hemoptysis, hematemesis, hematuria, and enterorrhagia may sometimes occur. Extreme anemia and some edema, especially of the ankles, are common. The soreness of the gums renders mastication difficult. There are increasing weakness, palpitation of the heart, emaciation, mental depression, usually impairment of appetite, and constipation, although sometimes diarrhea is encountered. The joints may become swollen and painful. Fever is common when the disease is far advanced, but in the early stages a normal or subnormal range of temperature is encountered.

Diagnosis.—In the greater number of cases the diagnosis is easy. Many individuals are usually affected at the same time, and there is a history of improper food. In single cases the disease may be confounded with some of the arthritic varieties of purpura, but the previous history, and the rapid improvement when suitable food is taken, render the diagnosis easy.

Prognosis.—In the main the prognosis is favorable.

Treatment.—A sufficient quantity of anti-scorbutic food should always be kept on shipboard as a prophylactic measure. The treatment of this disease consists in the use of antiseptic mouth-washes, such as dilute carbolic acid solutions, permanganate of potash, and nitrate of silver. The diet should consist of plenty of fresh vegetables. Lemon-juice and bitter tonics are also of great use.

INFANTILE SCURVY.

Synonym.—Barlow's disease.

Etiology.—This disease most often appears in infants between the ages of nine months and fourteen months; rarely is it met with later than the second year of life. It is generally believed that the disease is due to improper feeding—the exclusive use of condensed milk and of various proprietary foods. Sometimes cows' milk and sterilized milk are said to be the cause of this disease. It occurs in the children of the well-to-do oftener than in the children of the poor, for the reason that proprietary articles of food are more generally used among the former. According to Barlow, the child that is being suckled at the breast never develops the disease.

Pathology.—Subperiosteal hemorrhages causing separation of the periosteum are found, the blood finding its way between the epiphysis and the shaft of the bone. The legs are most frequently affected; the bones of the arms, the scapula, and the lumbar vertebræ may also be the seat of hemorrhages; later, the long bones will reveal marked thickening. The mucous membrane of the gums also becomes spongy.

Symptoms.—The infant grows weak, irritable, fretful, and loses appetite. Upon being handled or when the extremities are moved, pain is produced. The lower limbs are motionless (pseudoparalysis), the child dreading the pain which is produced by movements. Ulceration of the gums, extravasation of blood into the tissues, and bleeding from the mucous membranes may be encountered. There is usually some fever, although the temperature is rarely above 102° F. Rickets and infantile scurvy may coexist. Diarrhea is most commonly met with.

Prognosis.—As a rule, recovery is prompt, unless the disease is very far advanced.

Treatment.—Proper articles of food should be substituted for any of the proprietary articles. Fresh cows' milk and

meat-juice should be given in proper amounts. Orange- or lemon-juice is also beneficial. Proper hygienic surroundings and change of climate are indicated.

PURPURA.

Definition.—A condition, occurring in many diseases, characterized by extravasation of blood into the skin, mucous membranes, and internal organs, and sometimes by free hemorrhage from mucous membranes.

The alterations in the composition of the blood in purpura are, generally speaking, those of symptomatic anemia from hemorrhage.

Synonym.—Hemorrhagic diathesis (this term includes hemophilia).

Varieties.—(1) *Symptomatic purpura;* (2) *arthritic purpura;* (3) *purpura hæmorrhagica.*

SYMPTOMATIC PURPURA.

Infectious.—This is due to a variety of infective diseases, such as pyemia, septicemia, typhus fever, smallpox, scarlet fever, malaria, measles, and infectious endocarditis. The extravasation of the blood may be quite large, when it is called an ecchymosis; or pin-points, when it is called petechial.

Toxic.—Purpura may result from the administration of certain drugs, such as potassium iodid, ergot, mercury, belladonna, phosphorus, salicylic acid, and quinin. It has also been noticed from the virus of snakes.

Cachectic.—Various constitutional affections produce purpuric eruptions, such as leukemia, pernicious anemia, tuberculosis, Hodgkin's disease, Bright's disease, scurvy, and cancer. This may also be sometimes noticed in advancing years. The eruption is confined to the extremities, the wrists, the hands, and the legs.

Neurotic.—This occurs from lesions of the spinal cord, such as transverse myelitis, locomotor ataxia, and sometimes in hysteria and neuralgia.

Mechanical.—This form results from trauma, and in asthma, whooping-cough, and epilepsy.

ARTHRITIC PURPURA.

Under this term is considered a form of purpura which involves the joints and is sometimes spoken of as rheumatic. There are, however, no evidences of rheumatic manifestations.

Purpura Simplex.—This variety is most commonly met with in children. It is a mild condition, accompanied by purpuric spots upon the extremities, and sometimes upon the trunk and arms, with impairment of the appetite and diarrhea.

Purpura Rheumatica.—Synonyms.—Peliosis rheumatica; Schönlein's disease. (By many this condition is regarded as being rheumatic. It is sometimes preceded by sore throat.)

A condition which attacks both sexes with about equal frequency, most commonly in early life, between the ages of ten and forty. The joints, especially those of the lower extremities, are involved, many simultaneously. The disease is characterized by purpuric eruptions, which appear in crops, and by fever and articular pains. The eruption is commonly limited to the extremities and is bilateral; in severe cases the face and body may be involved. The fever is usually not high. Very rarely does it terminate fatally.

Diagnosis.—The diagnosis depends upon the involvement of the joints, many being affected simultaneously with purpura.

Henoch's Purpura.—It occurs most often in childhood, but sometimes in adults. A condition characterized by joint involvements, the joints being painful and swollen. Hemorrhages from the mucous membranes, gastro-intestinal disturbances, purpuric manifestations, and a tendency to relapse are also characteristic. It is most common in the young. The spleen is sometimes enlarged.

The prognosis is favorable.

PURPURA HAEMORRHAGICA.

Synonym.—Morbus maculosus Werlhofii.

This condition is frequently met with in the young, females being more susceptible than males. It is characterized by severe hemorrhages from the mucous membranes and by cutaneous extravasation. There is marked weakness, and, as a result of the hemorrhage,—which may take place from the respiratory mucous membranes, from the genito-urinary tract, or from the gastro-intestinal mucous membrane,—secondary anemia develops, which is frequently pronounced, and

death may result from loss of blood. Fever is generally present.

Hemorrhages into the skin are usually pronounced, being ecchymotic as well as petechial.

Diagnosis.—The diagnosis depends upon the rapid onset, the profound hemorrhage, the purpuric manifestations, and rapidly developing anemia. The condition may or may not be accompanied by fever.

Scurvy is diagnosticated from this condition by the presence of swelling and by tenderness of the gums and the history of the onset.

Treatment.—The cause, if possible, should be removed when dealing with symptomatic purpura. Tonics, such as arsenic, in the form of Fowler's solution, in ascending doses until the physiologic limit is reached, may be useful.

In dealing with free hemorrhages, such as are met with in purpura hæmorrhagica, the following ways may be of use in controlling it: Rest, compression, cold, ergot, tannic and gallic acids, acetate of lead, calcium chlorid, gelatin or collodion applied to the bleeding surface, or extract of suprarenal capsule. An attempt should be made to increase the coagulability of the blood and to restore it to its normal condition. If the hemorrhage is from a free surface that is easy of access, as the mucous membrane of the mouth or nose, the application of normal blood to the bleeding surface may be found of use.

HEMOPHILIA.

Definition.—A disease characterized by a tendency to hemorrhage, which is often uncontrollable, and is due to a deficiency in the coagulability of the blood. The coagulation is retarded, and frequently in this condition the blood does not coagulate in less than from thirty to fifty minutes.

Etiology.—There is a marked hereditary tendency, the disease being transmitted through the mother (who is rarely a bleeder herself, but the daughter of one) to the males of the family. The disease is not transmitted through the male offspring. Males are very susceptible, in the proportion of 13 to 1, and by some it is believed that the disease does not affect females. The condition manifests itself most frequently in early life, but sometimes not until early adult or middle life.

Pathology.—Coagulation of the blood is delayed. The bleeding, when it occurs, is of a capillary nature, there being constant oozing. It may take place from a free surface or into the tissues.

Symptoms.—The bleeding frequently results from trifling wounds, scratches, extraction of a tooth, or slight operations; or, again, it may occur without trauma from the mucous membranes or into the tissues. The most common form of bleeding is from the nasal mucous membrane (epistaxis). It also occurs from the mouth, stomach, lungs, kidneys, and urethra, and from various parts of the skin. Bleeding frequently takes place into the serous sacs, especially about the joints. Death may follow in a few hours or the hemorrhage may be prolonged over a period of weeks; in the latter instance the symptoms depending upon anemia manifest themselves.

Diagnosis.—The diagnosis depends upon the occurrence of uncontrollable bleeding in males,—either spontaneous or traumatic, the hemorrhage frequently recurring,—the history of the disease in some members of the family, and the peculiar mode of transmission.

Prognosis.—As age advances the prognosis becomes more favorable. There are some instances in which the tendency is outlived.

Treatment.—Prophylaxis.—Wounds should be avoided as much as possible, such as surgical operations, and it should also be remembered that the common practice of routine blood examination will be interfered with in this disease, and it is a good rule, before making a blood examination, *always to ask whether the person is a "bleeder" or if there is a family history of such a disease.*

When the bleeding is from a free surface and easy of access, compression and bodily rest may be tried. The local application of ice is often of value. Calcium chlorid and perchlorid of iron are recommended by many. Gelatin, collodion, and extract of suprarenal capsule may be found useful, being applied directly to the bleeding surface. Freshly drawn blood from a healthy individual may be employed as an application. The secondary anemia which follows should be treated with iron and arsenic.

MYXEDEMA.

Definition. — A disease characterized by myxomatous change of the subcutaneous tissues, due to pathologic lesions in the thyroid gland causing diminished or absent secretion.

Synonym.—Athyrea.

Varieties.—Three varieties of this disease have been found: (*a*) Myxedema, or adult myxedema; (*b*) sporadic cretinism; (*c*) operative myxedema, or cachexia strumipriva.

ADULT MYXEDEMA.

Etiology.—The disease is common in England, also in certain parts of Switzerland, but is less frequently found in America and other parts of the world. The disease occurs more frequently in women than in men, in the proportion of 6 to 1. Pregnancy seems to predispose, and it is more common in married women, especially those who have borne children. The disease is most often encountered between the ages of thirty-five and forty-five. Heredity plays a very slight part. Exophthalmic goiter seems to bear some relation to the disease, as individuals suffering from myxedema frequently have brothers or sisters who suffer from exophthalmic goiter. Exposure, mental strain, and hemorrhage also seem to predispose.

Pathology.—The thyroid gland shows constant lesions, in many instances being degenerated or atrophied. The gland is usually smaller than normal, and occasionally its congenital absence has been noticed. In some cases it is larger, but the secreting structure is atrophied, the increase in size being due to hyperplasia of fibrous tissue.

Extirpation of the thyroid gland in animals has produced symptoms that are identical with, or that closely resemble, myxedema in the human subject. The myxomatous changes affect the skin and its appendages; which are secondary to the lesion in the thyroid gland, and probably result from the diminished function of this organ, the internal secretion being diminished or absent. Sclerotic changes have been observed in the blood-vessels and the kidneys.

Bramwell clearly defines the physiology of the thyroid body as follows: "That the thyroid gland is in some manner or another (either directly or indirectly) concerned in the regulation of the metabolism of mucin or of substances which

form mucin, or that it is concerned in separating from the blood some substance or substances which either directly or indirectly (possibly through the nervous system) favor the production of mucin in the tissues," which, he says, also seems proved by the fact that large quantities of mucus are excreted by the kidneys as a result of active thyroid treatment in cases of myxedema. Extirpation of the thyroid gland in animals causes an enormous increase of mucin in the tissues in some instances.

Symptoms.—The onset is slow and the course of the disease is chronic. Changes which result from myxomatous degeneration of the skin and subcutaneous structures are often the first to attract the attention of the patient. The face becomes full, coarser, and round (moon-shaped), and the countenance is dull. The tissues around the orbit are swollen, and may suggest Bright's disease, but it will be noticed that the upper lid suffers as much as the lower. The lips present a bluish or purplish color. The nose becomes broader, the lips thicker, and the cheeks puffy. The ears are often swollen, the tongue is thickened, and the hair is scanty. Baldness not infrequently results. The skin sometimes shows a yellowish discoloration, being most marked about the exposed parts, and is quite resistant upon palpation. It is usually dry and brittle; the secretion of sweat is diminished and the color of the hair is changed. The teeth and nails are frequently diseased; the former being carious and the latter brittle. The body and neck increase in bulk, as do also the extremities, especially the hands and feet. Occasionally there is an increase in the saliva. Mental impairment develops as the disease progresses, as does also loss of physical activity. The gait becomes changed, being clumsy and sluggish. Articulation is impaired, speech being slow. Mental disturbance in some cases is pronounced, the patient developing either melancholia or mania. The tactile sense, as well as other special senses,—sight, hearing, taste, and smell,—is occasionally impaired.

The temperature is frequently subnormal and the patient experiences a sensation of cold. Secondary anemia results; this, however, not being marked.

The heart's action is slow and feeble. The urine may show an increase of mucin when thyroid treatment is instituted.

SPORADIC CRETINISM.

This condition occurs in the young between the ages of two and four years, the symptoms resembling closely those of adult myxedema. They are frequently ushered in by some one of the infectious diseases, notably scarlet fever or measles. Fully developed cases of sporadic cretinism are typical in their appearance. The body is dwarf-like; although young in years, the patients affected with this disease appear quite old. The mental, physical, and sexual developments are much interfered with. The features are heavy, the face is broad and coarse-looking, the body is thick-set, the abdomen is frequently pendulous, the hands and feet are swollen, the hair is usually abundant, but is straight and coarse, and the skin is dry, rough, and often scaly.

The thyroid gland shows the changes met with in adult myxedema, and in some cases is absent. The sexual organs are frequently underdeveloped.

OPERATIVE MYXEDEMA.

This results from the removal of part, or more commonly the whole, of the thyroid gland. The symptoms are similar to those already set forth in adult myxedema, and arise gradually.

Diagnosis.—The disease can rarely be mistaken for any other condition. The physical and mental changes are peculiar and highly characteristic.

Prognosis.—The prognosis is favorable if thyroid treatment is instituted early, being especially so in operative myxedema.

Treatment.—Thyroid extract should be administered as early as possible, and should be continued until all the symptoms disappear, and then a prophylactic dose must be given regularly. The dose varies from ¼ of a grain to 4 grains once daily. It should be increased gradually; large doses frequently produce acute thyroidism, serious cardiac depression attending this condition. After the administration of this drug loss of weight soon occurs. The mental and physical condition begins to improve; the hair, which is frequently thin, becomes thick, and changes to its original color. The sweat-glands become active and the heart's action is improved. General tonics, such as iron, quinin, and strychnin, may prove of value.

EXOPHTHALMIC GOITER.

Definition.—A disease characterized by protrusion of the eyeballs, enlargement of the thyroid gland, tachycardia, and a tremor.

Synonyms.—Basedow's disease; Grave's disease; Parry's disease.

Etiology.—The disease affects all classes of society; it is, however, most frequent between the ages of fifteen and forty, being quite rare at the extremes of age. It is commoner in the female in the proportion of ten to one. It sometimes follows some of the infectious, and occasionally some of the nervous, diseases. Not infrequently it exists in several members of the same family. Hereditary tendency is a feature. The exciting cause is said to be some profound shock, mental disturbances, overfatigue, fright, or great physical effort.

Pathology.—The protrusion of the eyeball is never so marked after death as it is antemortem. It is due to an excess of retro-orbital fat and to increased vascularity. The thyroid commonly shows a uniform enlargement; it is firm and of a brownish-red color. The chief feature is the increase in the secreting structure and marked vascularity. The thymus gland may be large. The heart shows hypertrophy and dilatation, or it may be normal. No characteristic lesions are found in the nervous system.

Symptoms.—The symptoms of the disease are probably due to some derangement of the nervous system as well as *to perverted or excessive secretion of the thyroid gland.* As stated in the etiology, it frequently follows grief, fright, shock, etc.

The early symptoms are irritability of temper, exophthalmos, and palpitation of the heart. The thyroid enlargement is not always noticed from the onset.

The protrusion of the eyeball often progresses with the thyroid enlargement. The eyes appear as though they bulged out of the head. Friends sometimes first inform the patient of this change. The protrusion may be so great as to render proper closure of the eyes during sleep impossible. Rarely only one eye protrudes (this commonly being accompanied by unilateral thyroid enlargement), or the protrusion of both may be very slight.

Von Graefe's Sign.—Upon lowering the visual plane, which may be accomplished by holding an object before

the patient and gradually lowering it, the upper lid fails to follow properly the eyeball downward—it lags behind.

Dalrymple's sign consists in a widening of the palpebral fissure, which appears to be due to spasmodic contraction of Müller's muscle.

Stellwag's Sign.—There is infrequent reflex winking, which may be ascribed either to the contraction of Müller's muscle or to some degree of anesthesia of the cornea. Dalrymple's sign is sometimes erroneously credited to Stellwag. Both v. Graefe's and Stellwag's signs have been noticed in other conditions.

Möbius' Sign.—This consists in the failure of convergence for near objects. This sign is not always present.

Vision, as a rule, is not affected. Painful spasm of the orbicularis palpebrarum and ulceration of the cornea are rare. Watering of the eyes is common.

The absence of constant dilatation of the pupil rather counts against involvement of the cervical sympathetic nerves.

The enlargement of the thyroid gland is not so extensive as is common in cystic goiter. It may be symmetric or asymmetric, and an accessory thyroid may be found, which is often enlarged. The gland presents pulsation, and on palpation a distinct thrill is felt. Upon auscultation usually a loud systolic murmur is noticed; sometimes even a double murmur. The protrusion of the eyes and the enlargement of the thyroid gland often develop simultaneously.

The heart action is rapid, varying from 90 to 160 a minute. The palpitation may be painful; the carotids pulsate visibly.

A muscular tremor affecting the whole body is found in nearly all cases. This tremor varies from eight to nine a second; it is usually bilateral, but in rare cases it may be unilateral, or may affect a single member.

Diarrhea and vomiting occur as prominent symptoms in many instances. Anemia and loss of weight usually develop.

Excessive sweating, sensations of heat and elevation of temperature, and marked flushings of the face, feet, hands, and body generally accompany the disease.

The urine is usually greatly increased in amount, and may contain albumin, and sometimes sugar. The respirations are accelerated; there may be a nervous cough. Neuralgia, headache, and mental alteration are not infrequent. Sleep is interfered with, the patient being restless; emaciation is marked, but the general health is not greatly impaired. The appetite

is good, sometimes excessive, and thirst is usually a marked symptom. The disease usually runs a chronic course; relapses are common.

Diagnosis.—The diagnosis depends upon the exophthalmos, the enlargement of the thyroid gland, tachycardia, and the tremor. It is attended with little difficulty.

Prognosis.—The disease may last for years, there is much uncertainty as to recovery, and, as before stated, relapses are common.

Treatment.—The ordinary house diet, well regulated, should be given. Hydrotherapy is often valuable. Rest in bed and ice-bags applied to the precordium give much relief to the patient; and as a result of this treatment the pulse-rate becomes slower and the patient receives much comfort. Digitalis, aconite, veratrum viride, tincture of strophanthus, and potassium bromid have been employed in this disease. Opium frequently gives great relief. Removal of the thyroid has been practised with but little benefit.

ACROMEGALY.

Definition.—A disease characterized by enlargement of the osseous structures, particularly the bones of the hands and feet, with constitutional symptoms.

Synonyms.—Pachyacria; megalacria; Marie's disease.

Etiology.—The disease is found in all races, commonly between the ages of fifteen and forty. The sexes suffer equally. Heredity seems to play a slight part in the predisposition, and cases are on record where several members of a family have been affected.

Pathology.—The pathology chiefly relates to the osseous system and to the pituitary body. The bones of the face become altered; the inferior maxilla is thickened and elongated, the cheek-bones are prominent, the nasal bones are thickened, the supraorbital regions are enlarged and prominent, and the head in general has a peculiar ape-like appearance. The anteroposterior diameter of the chest is increased, so that the thorax assumes a globular shape, the clavicle, ribs, and sternum are enlarged, the shoulders are rounded, and curvature of the spine develops (kyphosis and scoliosis). From this deformity an ape-like appearance is assumed. The vertebræ are thickened and the spinal processes are enlarged. The bones of the pelvis are also enlarged and thickened.

The long bones most commonly escape, the smaller bones being affected. The joints may be enlarged. The bones of the feet and hands are elongated and thickened, so that the breadth and length are increased. The wrists and the ankles may also be increased in size.

The pituitary body is found either enlarged, the seat of a new growth, or atrophied. It is believed that the disease of the pituitary body acts as the primary, the fundamental lesion, and produces either an arrested, an increased, or a perverted secretion; and this, in return, disturbed metabolism of the body. The thyroid gland is often found enlarged. In the progress of the disease the mammary glands, the ovaries, and the testicles are sometimes found atrophied (these glandular structures seem to bear a relationship to one another).

Symptoms.—The important symptoms of the affection are the enlargement of the hands and feet—those relating to the osseous system, already noted in the pathology. The skin is thickened and warty. The growth of the hair is often increased, becoming long and coarse. Headache is common and is usually referred to the eyeballs. There is often lassitude. Speech is thick and slow. The tongue is flabby and large. Blindness may occur from atrophy of the optic disc. Loss of smell and of taste are common, and hearing may be impaired. The menopause occurs before the normal period. Muscular weakness and excessive perspiration are common.

Prognosis.—The prognosis is unfavorable.

Treatment.—There is no remedy that is known to be of service in this disease. The drugs that have been used are iodid of potassium and thyroid extract.

PART VIII.

DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE NERVES.

NEURITIS.

Definition.—Neuritis is an inflammation of a nerve ; it may be either localized (that is, confined to a single nerve-trunk) or multiple (affecting a number of nerves).

LOCALIZED NEURITIS.

Etiology.—Cold is most frequently the cause. The affection is sometimes due to trauma, such as contusions, stabs, or cuts, or to stretching or tearing of a nerve, as might result from a fracture or dislocation. Pressure from muscular contraction may also cause this affection. It may result from extension, as from disease of the bone through which the nerve passes. It is often due to toxins and morbid states arising in the course of infectious and constitutional diseases. The mineral poisons are not infrequent causes. Alcohol most commonly produces peripheral neuritis.

Pathology.—The inflammation may be confined to the nerve sheath (perineurium), to the interstitial part, or to the axis-cylinder. In the first instance the nerve is particularly swollen, red, and infiltrated with numerous leukocytes ; in the last instance degenerative changes of the axis-cylinder are noticed (parenchymatous neuritis). The nuclei of the nerve-cells may consist of oily looking globules. The degeneration, according to Waller, extends down the nerve, because the fibers are cut off from the trophic cells. The muscles may undergo atrophy. Increased fibrous tissue may form in the nerve if recovery takes place.

Symptoms.—The constitutional disturbance is slight, and fever is rarely a symptom. Pain and tenderness are the principal symptoms, and are usually localized to the nerve-trunk and its distribution. The pain varies in intensity and character, and is described as burning, aching, boring, or shooting. Commonly it is aggravated at night, or in positions in which pressure upon the nerve-trunk may occur. The nerve-trunk may be swollen, and some reddening of the skin occasionally appears. Muscular twitching and contraction sometimes occur along the course of the nerve. If the disease be protracted, such changes as muscular weakness and wasting, slight edema, with a tremor, hyperesthesia, paresthesia, or anesthesia may develop. Acute cases usually end in recovery in about two weeks. Chronic cases may last for months or years, and may then gradually subside. The electric reaction is normal in mild cases; in severe cases the reaction of degeneration may take place.

Diagnosis.—The differential diagnosis between neuritis and neuralgia must be made. In neuritis the pain is continuous, and occurs along the course of the nerve. In neuralgia the pain is intermittent in character, and is often relieved by pressure, whereas the pain of neuritis is aggravated by pressure. Altered sensation is in favor of neuritis.

Prognosis.—The prognosis is favorable in mild cases. If the disease becomes chronic, it may last for months or years. Cases due to suppuration are less likely to recover.

Treatment.—It is important to remove the cause, if possible. Rest is essential. When it is possible to splint the part, this should be done. Applications of cold by means of an ice-bag are useful. In some cases heat is more desirable. The use of a blister along the nerve-trunk in severe cases is of benefit. If the pain is very severe, morphin hypodermically must be resorted to. Salicylate of sodium is of value in the forms due to cold and exposure. In the chronic variety electricity is of use, galvanism being preferable. The weakest current that can be appreciated is most efficient. When wasting takes place, massage should be tried.

BRACHIAL NEURITIS.

Definition.—Inflammation of the brachial plexus.

Etiology.—This occurs particularly in gouty individuals, affecting both sexes equally. The majority of cases arise after

the fiftieth year of life. Exposure to cold is said to be a predisposing factor.

Symptoms.—Pain in the course of the distribution of the brachial plexus is the prominent feature. It is frequently encountered in the wrist, in the axilla, above the clavicle, and in the scapular region. At first it is intermittent, and felt only upon certain movements. It is usually severe. As in other forms of neuritis, trophic changes may occur. The disease is not common.

Prognosis.—The disease lasts for months, sometimes for years, recovery rarely being complete. Relapses are extremely common.

Treatment.—The treatment is the same as in other forms of neuritis.

SCIATICA.

Definition.—Inflammation of the sciatic nerve.

Etiology.—The disease is more frequent in males than in females, in about the proportion of four to one. It is rare in children, and is most frequent between the ages of twenty and fifty. The gouty and rheumatic diatheses are predisposing causes. The exciting cause most often is exposure to cold; wet cold, it is said, being an especially favorable cause. Occasionally sciatica, it has been claimed, may be due to the poison of acute rheumatic fever. Pressure and intrapelvic disease may give rise to the affection.

Pathology.—The pathology is the same as that of other forms of neuritis.

Symptoms.—Pain along the course of the sciatic nerve,—which is commonly felt at the back part of the thigh, in the region corresponding to the sciatic notch,—behind the knee, and below the head of the fibula, is the most important symptom. The pain may be diffused and may extend from the sciatic notch to the toes. All muscular movements aggravate the pain. In long-standing cases trophic changes occur.

Prognosis.—The disease is liable to be obstinate, usually lasting for months.

Treatment.—Rest by means of splinting the limb is important. Attention should be directed to the cause of the disease. If of rheumatic origin, the salicylates are useful. The use of mercury has been advised by some authorities, notably Gowers. Phenacetin, antipyrin, and other members of the coal-tar group are useful; however, in severe cases morphin

is necessary. Injections of chloroform or sterile water into the tissues along the course of the sciatic nerve may give relief in severe cases. Surgical interference by nerve-stretching should be resorted to only when other methods have failed to give relief.

MULTIPLE NEURITIS.

Definition.—An inflammation involving many nerves, often by a symmetric change. These may be affected simultaneously or in rapid succession.

Synonyms.—Peripheral neuritis; polyneuritis.

Etiology.—Multiple neuritis is almost invariably caused by some toxic agent, the most frequent, with the exception of lead and diphtheria, being alcohol. It occurs most often in persons who use strong liquors, but malt liquors also give rise to the condition. The disease is said to be more prevalent in females than in males. Scarce and improper food and exposure to cold are asserted to be predisposing factors. The fumes of some gases, such as carbon monoxid from charcoal stoves, and the fumes of anilin and bisulphid of carbon, are said to cause multiple neuritis through inhalation. The condition is frequently a sequel of infectious diseases; however, it more frequently follows diphtheria than any of the others. It takes place as a sequel in enteric fever, and more rarely from measles, pneumonia, influenza, scarlet fever, variola, varicella, and erysipelas. It is exceedingly uncommon after septic infection and syphilis. Neuritis occasionally occurs in the course of tuberculosis, but is very frequent in the course of leprosy. Of the metallic poisons, lead is the most frequent cause, but arsenic and mercury are also occasional factors. It occurs in the cachexias, such as malignant disease and anemia, and the affection has been noticed in the puerperal state. It is sometimes met with in the aged, arterial sclerosis probably being the exciting cause. Perforating ulcer and Raynaud's rheumatoid arthritis have been attributed to peripheral neuritis.

Pathology.—The pathology of multiple neuritis is practically the same as that of neuritis occurring in isolated nerves. The secondary changes which occur in the muscles are quite pronounced. Changes in the spinal cord, such as meningitis or chronic myelitis, may be encountered. Vacuolation of the ganglion cells and atrophy of the gray matter sometimes accompany multiple neuritis.

Symptoms.—The symptoms vary. According to Gowers, three distinct varieties must be differentiated as to the prominence of certain symptoms, such as the motor and sensory phenomena, or those of incoordination. Pure types of each class may exist, but it is much more usual to find them combined. The disease mostly begins abruptly, but prodromes may exist, and may extend from a period of weeks to months, characterized by numbness and tingling in the hands and feet and by muscular cramps. In the acute cases the onset resembles the beginning of the acute infectious diseases. The temperature rises abruptly to 102° F. or 104° F., often with splenic enlargement, slight albuminuria, and even jaundice. Active pain in the limbs, with slight swellings at the articulations, is sometimes present, so that the attack may resemble acute rheumatic fever. Numbness and tingling, some pain, and muscular cramp, which occurs in the extremities, precede or accompany loss of power in certain muscle groups. This is an early phenomenon. The extensors of the hands and the anterior tibial group are the muscles chiefly involved; wrist- and foot-drop, as a result, are quite characteristic. Tenderness in the muscles, especially in alcoholic cases, aggravated by the least change of posture or by the slightest pressure, is a symptom of great importance. Paralysis may develop rapidly, and be complete in a few days. Occasionally the onset is more gradual, the paralysis requiring several weeks before it occurs. Either the arms or the legs may be involved, but the paralysis is always symmetric. It is most common for both arms and both legs to be affected, the lower extremity being more frequently involved than the upper. The muscles primarily involved are those at the periphery of the part, as below the elbow and knee, principally the groups supplied by the musculospiral and popliteal nerves. From a loss of power in the muscles of the anterior surface of the leg the "*steppage*" gait, in which the thighs are unduly flexed, results. The muscles of the neck, back, chest, and abdomen are rarely involved, save in the severest cases. When paralysis of the muscles of respiration or of the diaphragm takes place, the attack is liable to terminate fatally. Increased cardiac action and paralysis of the larynx occur from involvement of the pneumogastric nerve. Unless the spinal cord is affected the sphincters are not involved. Only in neuritis due to diphtheria have the cranial nerves been found affected; however, in rare instances there may be paralysis of the

third, fourth, and sixth nerves. Sooner or later nutrition of the muscles suffers. They become wasted and flabby, and deformities from contractures arise. Sensory involvement has already been indicated. It consists in numbness, tingling, and a pricking sensation, as of pins and needles, especially in the hands and feet. Fibrillary tremor is sometimes present in the affected muscles. Incoordination is not infrequent. There is difficulty in employing the hands for delicate manipulations, and a decided unsteadiness of gait is noticeable. The resemblance to locomotor ataxia is occasionally marked. The knee-jerks are absent in severe cases. There is often slight edema of the ankles and hands. Glossy skin, loss of hair, and trophic changes in the nails are not uncommon.

Electric Changes.—The degree of irritability of nerve and impaired contractility of the muscle are of importance, both for diagnosis and prognosis. From secondary changes in the muscles it may be necessary to employ a strong galvanic current.

Complications.—Pulmonary complications, especially when the respiratory muscles are affected, are frequent, and may be the cause of death. Often in the alcoholic variety there is pulmonary tuberculosis.

Prognosis.—The severer cases may prove fatal in one or two weeks from paralysis of the respiratory muscles, but this is rare. The disease is most often prolonged; recovery is gradual, and may extend over a period of months or years. The muscular power returns slowly. Complete recovery, however, is the rule, even when marked paralysis has occurred, and has been prolonged for more than a year.

The Alcoholic Variety.—This is more frequent in women, in whom it is exceedingly difficult to get a history. The premonitory symptoms consist of cramps, numbness, and slight incoordination. The paralysis takes place most commonly in the lower extremity, later showing itself in the upper extremity. The symptoms are even accompanied by severe colic resembling visceral neuralgia. The pulse is rapid, weak, and irregular, and vomiting, due to the gastric disturbance, is frequent. Mental symptoms such as occur in delirium tremens are common. There is loss of memory, especially for recent events, the patient becoming suspicious of attendants or friends, and showing various delusions. Convulsions are rare.

Occasionally recurrence takes place, the interval between the attacks varying from a few months to several years.

Treatment.—It is important to ascertain the cause, which should be removed if possible. In the case of alcohol, a trustworthy nurse should be employed or the patient should be treated in the hospital. Absolute rest in bed is important. When the pain is severe, splinting, if practicable, should be employed. Hot or cold applications, whichever are most grateful to the patient, may be used. If the pain is severe, morphin may have to be resorted to. Care should be taken to prevent contractures, which is best done by the use of splints. When cold has been the causative factor, the salicylates are of value. Iron and cod-liver oil are important as tonics. Massage and electricity should be employed in the later stages of the disease. A weak galvanic current may relieve pain, the positive pole being passed over the painful muscles and nerves.

ENDEMIC NEURITIS.

Synonyms.—Beriberi; kakké.

Etiology.—This disease is prevalent in parts of Japan, in the Philippines, in India, and along the coast of Brazil. It is believed to be due to a special micro-organism. This view, however, is not generally accepted. It is supposed that the disease may be acquired by contagion. It has been thought that a diet of decayed fish or spoiled vegetables, or a diet of certain kinds of mussels, may produce the affection. Other observers have asserted that the disease is transmitted through drinking-water.

Symptoms.—The disease usually begins with a sense of heaviness in the extremities, the muscles tiring easily, perverted sensation, and with great irritability of the heart. There is fever, edema, rarely anasarca. The edema shows itself first in the legs, and is quite constant. The urine is scanty; albumin is not often present. An increase in the amount of urine shows improvement in the patient's condition. There is an early change in the electric reaction, particularly of the peroneal nerves, showing a slight reaction of degeneration.

The mortality varies from 3% to 60% or 70%, as the fatal cases are those in which there is involvement of the phrenic or pneumogastric nerve.

Treatment.—The treatment consists primarily in the removal of any and all causative factors, and then in treating the

disease as in other forms of neuritis. On account of the great cardiac asthenia, heart tonics are necessary, as caffein, strychnin, and alcohol. A mild purge at the onset is beneficial.

DISEASES OF THE CRANIAL NERVES.

DISEASES OF THE OLFACTORY NERVE.

Anosmia (loss of smell) is most often due to local trouble in the nasal mucosa. If the olfactory bulbs are damaged by tumors, syphilitic lesions, or injury, loss of smell may occur.

Treatment.—The treatment depends upon the cause. Strychnin, which experimentally stimulates the olfactory nerve, should be tried.

DISEASES OF THE OPTIC NERVE.

Etiology.—This disease may occur in the optic nerve, in the chiasm, or in the tract. These parts may be damaged by tumors, syphilitic lesions, hemorrhage, or meningitis.

Optic neuritis is usually a symptom of grave disease, resulting especially from cerebral tumors or abscesses.

Symptoms.—If the neuritis be complete, blindness will occur upon the affected side. The pupillary reaction is absent, and atrophy can be detected in a few weeks by the use of the ophthalmoscope. If the damage be partial, narrowing of the field of vision occurs. Disease of the optic chiasm, when complete, causes blindness of both eyes and total loss of the pupil reflexes to light. Lesions in the situation of the optic tract, between the optic center and the chiasm, are accompanied by heteronymous hemianopia. Only those lesions due to syphilis are amenable to treatment.

OPTIC NEURITIS.

Etiology.—In rare instances this may be due to exposure to cold and wet. The majority of cases, however, are symptomatic of cerebral disease. Gowers and Bramwell claim that 80% of the cases are associated with cerebral tumors. It occurs in the course of some of the infectious diseases, such as enteric fever, scarlet fever, measles, and influenza. It takes place in diabetes, leukemia, and Bright's disease. The disease may arise in several members of a family, the females most often escaping.

Pathology.—The optic nerve reveals interstitial neuritis. The entire nerve or only parts of it may be affected. Swelling of the papilla is noticed, with haziness of the margins, the condition being known as papillitis (choked disc). In some rare instances edema is also added, so that the papilla becomes very prominent and assumes the shape of a mushroom. In rare instances optic neuritis may exist without the choked disc. It may be secondary to inflammation of the brain membranes.

Symptoms.—In the severest cases early symptoms may be entirely absent, in which case the disease can be recognized only by means of the ophthalmoscope. Vision may be but little affected; usually there is some contraction of the visual field.

Prognosis.—Mild cases may recover, but even these frequently terminate with some loss of vision. Severe cases almost invariably give rise to total blindness.

Treatment.—The treatment is symptomatic. If there be evidences of syphilis, mercury and iodid of potassium are indicated. Tobacco and alcohol must be prohibited.

PARALYSIS OF THE OCULAR NERVES.

The muscles of the eyeball are supplied by the third, fourth, and sixth nerves. Affections of these nerves belong to the domain of ophthalmology.

DISEASES OF THE FIFTH NERVE (TRIFACIAL).

The fifth nerve is the nerve of sensation of the face and the anterior part of the scalp. It consists of three main trunks: The *ophthalmic division* supplies the orbit, the lacrimal glands, the skin of the forehead and scalp, the tip of the nose, and the anterior part of the nasal mucous membrane. The *superior maxillary division* supplies the skin over the malar bone, the root of the nose, the infraorbital region, the upper lip and the greater part of the nasal mucous membrane, the palate, the upper part of the pharynx, and the teeth in the upper jaw. The *inferior maxillary division* supplies the skin in the temporal region, lower lip, chin, parts of the ear, and external auditory meatus, and also the mucous membrane of the mouth, the tongue, and the lower teeth. The motor division of the nerve innervates the muscles of mastication.

Paralysis of the Motor Portion.—Parts of a nerve or the entire nerve may be affected. Paralysis is usually due to deep-

seated disease, as the nerve is rarely affected peripherally. It may arise from pressure from tumors, meningitis, disease of the bone, or syphilis.

Symptoms.—The important symptoms are severe pain along the course of the distribution of the nerve, accompanied by anesthesia or hyperesthesia. Paralysis of the muscles of the jaw may occur. Atrophy of the muscles of the face may result.

Treatment.—Treatment should consist in the use of iodid of potassium in large doses. If the pain be severe, opium in some form will have to be resorted to. Galvanism and faradism are of use in some cases.

DISEASES OF THE SEVENTH NERVE (FACIAL PARALYSIS; BELL'S PALSY).

Etiology.—From the exposed position of this nerve neuritis due to cold and exposure frequently takes place. Disease of the middle ear may also give rise to injury of the nerve. Traumatism is responsible in some instances. It may be due to lesions about the facial nucleus.

Symptoms.—The appearance of the patient is characteristic. Expression has changed upon the affected side of the face, which is immobile. The furrows upon the face and forehead are smoother than upon the unaffected side. The labionasal fold has disappeared, the mouth being drawn toward the healthy side, with the angle of the mouth depressed. The eye is open, owing to paralysis of the orbicularis palpebrarum. There is inability to move the lid, also to use the facial muscles to laugh or smile upon the affected side, the mouth being drawn toward the unaffected side. The patient can not wrinkle the forehead. The eye remains open during sleep. The patient can not whisper, blow out a candle, or expectorate properly, and saliva is constantly dribbling from the mouth. Speech may be affected. If the nerve be damaged between the geniculate ganglion and the region of the chorda tympani, taste is lost upon the anterior two-thirds of the tongue. There may be abnormal sensitiveness to sound. Spasmodic twitching in the paralyzed muscles occurs late, and secondary contracture may take place in severe cases. In mild cases electric reaction shows diminished irritability, or in very mild cases no electric changes may be apparent. In severe cases irritability is completely lost to both currents.

Prognosis.—If there be complete reaction of degeneration,

recovery can not be expected for several months. The majority of cases, however, recover completely.

Treatment.—Cases due to cold and exposure are best treated by large doses of the salicylates; syphilitic cases, by the use of iodid of potassium in large doses. Warm fomentations are of use in many cases. In the severer cases a blister should be applied behind the ear. In case of severe pain, antipyrin and phenacetin are of value. The application of weak galvanic currents preserves the nutrition of the muscles and hastens recovery.

DISEASES OF THE AUDITORY NERVE.

Symptoms of the affection of this nerve consist in impairment of hearing or complete deafness. Disease in the labyrinth is the most frequent cause. It may be due to acute or chronic inflammation, spreading from the tympanum, to syphilis, or, in old persons, to degenerative changes. It may result from tumors and meningitis. Atrophy may occur in tabes, and may appear in elderly persons.

DISEASES OF THE GLOSSOPHARYNGEAL NERVE.

Very little is known of diseases of this nerve, owing to its numerous connections and absence of isolated lesions. It is most likely that paralysis of the nerve gives rise to difficulty in swallowing, and to loss of sensation in the roof and walls of the pharynx. It has as yet not been determined whether the glossopharyngeal nerve is a special nerve of sense or not.

DISEASES OF THE PNEUMOGASTRIC OR VAGUS NERVE.

This nerve has a long course, being distributed to the pharynx, esophagus, larynx, lungs, heart, stomach, spleen, and intestines. Paralysis may occur from disturbance within the skull, such as injury, and from pressure by morbid growths, meningitis, or aneurysm. The nucleus of the nerve may suffer from inflammatory changes. Causes outside of the skull may be due to surgical operations in dividing the nerve or to compression by tumors.

Symptoms.—Division of the nerve in animals is followed by an increase of cardiac action, while the respiration becomes slower and more pronounced. The same symptoms occur when the nerve is divided in man from surgical operations or

from injuries. Paralysis of the vocal cords is likely to occur. There is difficulty in swallowing and vomiting is frequent.

Treatment.—The treatment is symptomatic.

DISEASES OF THE SPINAL ACCESSORY NERVE.

Diseases similar to those which give rise to disease of the pneumogastric also affect the spinal accessory. When the external part of the nerve is diseased, paralysis of the sterno-cleidomastoid and upper part of the trapezius occurs. This is followed by wasting.

Treatment.—The treatment is symptomatic.

DISEASES OF THE HYPOGLOSSAL NERVE.

This is purely a motor nerve, supplying the muscles of the tongue. Paralysis occurs from cerebral lesions involving the fibers of the nerve from the cortex to its nucleus. Lesions of the nucleus are most often bilateral. Locomotor ataxia and syringomyelia give rise to unilateral disease. Peripheral paralysis is extremely uncommon.

NEURALGIA.

Definitions.—Neuralgia is a term used to denote pain in the course of a nerve, unattended by structural change. The border-line between neuritis and neuralgia can not be too closely drawn, but the term neuralgia should be used to express pain unattended by motor or trophic changes.

Etiology.—Neuralgia is particularly a disease of middle life. It is rare in childhood and in old age, and occurs more frequently in men than in women. Some females show a hereditary tendency to neuralgia, and it is frequent in neurotic females. It is common in debilitated and anemic individuals, frequently accompanying overwork and worry. The disease is often unilateral, and when so, the left side is more frequently affected than the right. Cold is a common exciting cause, as are malaria and gout. Irritations of various kinds, such as a carious tooth, may produce neuralgia. Eye-strain is a predisposing cause.

Symptoms.—Pain is the most important symptom. It shows a marked tendency to periodicity, and is paroxysmal in nature, being described as burning, shooting, or darting in character, not increased by motion, and often relieved by

slight pressure or friction. There may be increased secretion of saliva and tears, and a slight elevation of temperature. Muscular twitchings may occur. The duration of an attack varies from an hour to a day or more. Neuralgias show a constant tendency to recur at irregular intervals.

Neuralgia is very common along the course of the fifth nerve, when it has been called *tic douloureux*. It may involve the branches of the fifth nerve, and when it involves the ophthalmic branch, a tender point at the extremity of the nerve—the supraorbital notch—is indicated by the patient. When it involves the second branch, the infraorbital region is painful, and in the inferior maxillary division the tender point is in the region of the zygomatic arch.

Other forms of neuralgia are known as *cervico-occipital*, *brachial*, *neuralgia of the phrenic nerve* (which is exceedingly rare), *intercostal neuralgia*, *lumbo-abdominal neuralgia*, *visceral neuralgia*, *sacral neuralgia*, *coccygodynia*, and *neuralgia of the feet*. By the term reflex neuralgia is meant pain due to disease in organs distant from the actual seat of pain. This may occur in diseases of the eye, ear, nose, and throat, in diseases of the stomach and liver, or in uterine disease.

Prognosis.—The prognosis as regards recovery is good, although the disease is extremely stubborn, and shows a constant tendency to recur.

Treatment.—It is especially important to treat the underlying condition: thus, if the disease be reflex, it is important to give attention to the affection giving rise to it; if it be anemia or malaria, these causes must be given full attention. Hygienic treatment is of importance. Change of scene and residence, with good nourishing diet, are necessary. Many drugs have been recommended for the treatment of the paroxysm—quinin, the coal-tar analgesics (such as phenacetin), antifebrin, and so on. A combination of phenacetin and caffein, or a combination of phenacetin and salicylate of sodium, may be of use. Morphin should be avoided if possible, as there is great danger of the patient acquiring the opium habit. Aconitin, gelsemium, and belladonna are valuable. Local treatment is sometimes of use; thus, hot and cold applications, liniments containing menthol, and occasionally electricity may be tried. In protracted cases surgical interference should be thought of. Often surgery has rendered brilliant results.

DISEASES OF THE SPINAL CORD.

ACUTE SPINAL MENINGITIS.

Definition.—This term includes the various acute inflammations which affect the spinal pia mater or dura mater. If the inflammatory condition involve chiefly the pia mater, the process is diffuse, and extends often to the internal surface of the dura mater. A separate affection of the dura, as in acute purulent inflammation, occurs only secondary to disease of the bone or from trauma.

Synonyms.—Acute spinal leptomeningitis; acute internal meningitis.

Etiology.—The disease is always due to infection from micro-organisms, and most frequently the micro-organisms are of the pyogenic group. In tuberculosis in which disease of the cerebral membranes also occurs the spinal membranes are likely to be affected. Cerebrospinal fever, an affection of the membrane of the cord, is almost constantly associated with inflammation of the membranes of the brain. The affection arises in the course of the acute infectious fevers, such as croupous pneumonia, scarlet fever, enteric fever, and small-pox, and in the course of pyemia and septicemia. The disease may result from direct or indirect injury to the spinal column, from fractures or wounds, or from operation on the vertebral column. This affords a ready access to bacteria causing infection. Acquired syphilis of recent origin may give rise to an acute inflammation of the membranes of the cord, but this is rare, the process being much more likely to be subacute—that is, nonsuppurative. The affection appears more commonly in men than in women, and is more frequent before early adult life than after this period. Exposure to cold and wet, traumatism, and overexertion may be said to be predisposing causes.

Pathology.—In the first stage of the disease the internal membranes are hyperemic; later, an exudate forms, which may be fibrinous, semisolid, fluid, pus-like, or purulent. Microscopically, numerous leukocytes, a few red blood-cells, and fibrin are noted in the exudate. The inflammatory process may spread to the nerve-roots. When the pathologic process is of a tubercular nature, a gelatinous exudate is encountered, and

tubercles are noticed. The cord may be invaded by the pus, producing myelitis.

Symptoms.—As the lesions are rarely entirely spinal, and as parts of the cerebral meninges are also involved, the purely spinal symptoms are likely to be masked by the cerebral symptoms. It is, therefore, rare that the symptoms should alone be associated with cord phenomena. The disease usually begins with well-marked chills and a temperature which is of the septic type. There is severe pain in the back, increased by motion, which radiates into the upper and lower extremities. Rigidity of the muscles of the back, and sometimes opisthotonos, occurs. Tonic spasm in the muscles of the extremities and of the abdomen and chest is prominent. Occasionally tonic spasm gives place to clonic spasm. Hyperesthesia is general and marked, the reflexes are exaggerated, and ankle-clonus is pronounced. There may be retention of urine and feces from paralysis of the sphincters. Kernig's sign may be present. (See page 251.) If the acute symptoms subside, paralyses show themselves, paraplegia being the most common form. The reflexes become normal, and finally disappear, so that loss of knee-jerks occurs late in the disease. In severe cases death may take place in a few days. In the majority of instances the duration is from one to two weeks. Even in cases in which recovery occurs, rigidity and weakness of the muscles may last for months or years.

Diagnosis.—The direct diagnosis depends upon the severe pain in the back, radiating to the upper and lower extremities, and upon the rigidity of the muscles, the hyperesthesia, and the acute febrile course, with septic phenomena.

Prognosis.—Danger to life is imminent, and it is usually proportionate to the severity of the symptoms and the gravity of the fever. Traumatic cases are most liable to recover, especially when the affection occurs in persons in middle life; however, the prognosis must be put down as exceedingly serious.

Treatment.—Absolute rest in bed is important. If the patient can be induced to lie upon his side, it is better than to have him rest upon the back. Mild laxatives are useful. Severe pain should be counteracted by the use of opiates. Dry or wet cups along the spine are useful, followed by the application of ice-bags. Gowers favors mercurial inunctions. For the contractures and pain which arise during the course of the disease, hot baths and hot douches are of use. If there

should be bone disease with pus formation, surgical interference is necessary.

CHRONIC SPINAL MENINGITIS.

Definition.—A chronic inflammatory affection of the internal surface of the dura mater or of the pia and arachnoid of the spinal cord.

Synonyms.—Chronic leptomeningitis; chronic internal pachymeningitis.

Etiology.—Chronic spinal meningitis is frequently due to syphilis. The form due to disease of adjacent bone, which is usually of tubercular nature, is the variety that will be described here. It may occasionally be primary, although this is rare, when exertion and exposure to cold are said to favor its development. It may result from severe concussion of the spine. It most commonly follows disease of the bone, such as caries, sarcoma, or carcinoma affecting the vertebral column. It may also follow the acute variety.

Pathology.—The process may be localized or quite general. In mild cases the internal membranes are simply opaque and somewhat granular. In advanced cases the membranes are thickened, owing to formation of fibrous connective tissue, and the blood-vessels of the pia also show fibroid thickening of their coats. The nerve-cells at the periphery of the cord frequently undergo degenerative changes and become atrophied. The nerve-roots may also be thickened and compressed. Hemorrhages may occur in the membranes. Degenerative changes, both ascending and descending, are frequently observed. The cerebrospinal fluid may be increased in density.

Symptoms.—The symptoms vary, depending upon the amount of the spinal membrane involved. They usually consist in pain in the back, increased by movement; stiffness of the muscles; and, if the cervical region be involved, retraction of the head. Pain upon pressure over the vertebral column, which may radiate to the upper or lower extremities, is a symptom. Slight tremor, and occasionally clonic spasm in one or more extremities, combined with areas of hyperesthesia, are noticed. After a longer or shorter interval, of weeks or months, paralytic phenomena appear, with loss of sensation, the muscles being wasted, and reactions of degeneration manifest themselves.

Diagnosis.—The diagnosis depends upon the gradual oncome of the symptoms just enumerated, without fever.

Prognosis.—The prognosis varies. Danger to life arises from the impaired health due to prolonged disability. In some cases, through treatment, the irritative symptoms disappear, and partial return to health occurs. The prognosis is most favorable in traumatic cases in which the lesion is small in extent, and in which there is the possibility of surgical interference.

Treatment.—Rest in bed is of the greatest importance. Counterirritation by a mild application of the Paquelin cautery is of use. It relieves pain and aids absorption of certain inflammatory products. Hot baths and hot douches to the spine are of great benefit in relieving pain. Iodid of potassium and mercurial inunctions must be tried in syphilitic cases. Opiates should be resorted to only when the pain can be relieved in no other way. Massage and galvanism are of value when muscle wasting begins. The general hygiene of the patient must be looked after.

HYPERTROPHIC CERVICAL PACHYMENINGITIS.

Definition.—A chronic inflammation of the dura mater, affecting particularly the inner layer, involving also parts of the pia and arachnoid, which may lead to decided thickening of the dura mater of the cord, producing irritation and compression of the nerve-roots and of the substance of the cord in the cervical region.

Etiology.—The disease occurs more frequently in men than in women, between the ages of forty and fifty. The majority of cases are not of syphilitic origin. Local trauma and exposure to cold may be said to predispose. It is a very rare affection.

Pathology.—In this disease the dura mater becomes greatly thickened, owing to the hyperplasia of the fibrous connective-tissue. In some portions fully developed fibrous connective tissue is observed. The arteries may reveal thickening of the coats, or there may be hyaline degeneration. The dura may become closely adherent to the pia and arachnoid.

Symptoms.—The early symptoms consist of pain between the shoulders, in the back of the neck, and in the head, with slight rigidity of the muscles, tenderness over the cervical

vertebræ upon pressure, anesthesia and paresthesia in this region, and symmetric pain in the region of distribution of the ulnar and median nerves. These symptoms are due to irritation of the nerve-roots. The rigidity of the muscles may be accompanied by a coarse tremor. After a variable period, of weeks or months, the pains disappear, giving place to paralytic phenomena, due to compression of the nerve-roots. Partial or complete anesthesia takes place. Muscular atrophy in the region of the median and ulnar nerves, and reactions of degeneration, are prominent. The paralysis and wasting occur in the muscles of the hands, and contractions in the flexors of the fingers may lead to a peculiar deformity of the hand. Later, the disease may extend to other portions of the cord, and sensory symptoms below the lesions may arise. Occasionally the sphincters are affected. Peculiar speech may develop, known as the "staccato speech," resembling the form observed in multiple sclerosis. It is said to be due to embarrassed respiration. The pupillary reaction is sluggish. It is important to remember that the involvement of muscles is symmetric.

Diagnosis.—In typical cases the diagnosis is easy. Tumors of the cord may give rise to difficulty in diagnosis, but symptoms from pressure of tumors of the cord come on much more rapidly than in pachymeningitis. The disease should be diagnosticated only in the absence of disease of the bones of the vertebral column.

Prognosis.—The progress of the disease may extend over many years. In rare cases recovery is said to have occurred.

Treatment.—Attention should be given to the general hygiene of the patient and good nutrition should be maintained. Pain should be relieved. Warm baths are useful, and it has been said that the galvanic current gives relief.

HEMORRHAGE INTO THE SPINAL MEMBRANES.

Definition.—Hemorrhage within the dural sac, known as intrameningeal, or hemorrhage external to the dura, known as extrameningeal. The latter form is by far the more common.

Synonym.—Meningeal spinal apoplexy.

Etiology.—It is most often the result of injury, as from blows or falls. Some diseases accompanied by convulsions may lead to spinal hemorrhage from interference with the circulation of blood in the spinal canal. It may appear in the blood

dyscrasias and in the acute infectious diseases; also as a result of rupture of an aneurysm into the spinal canal, in which the vertebræ have been eroded. It may occur from the extravasation of blood from a cerebral hemorrhage into the membranes of the cord. It may sometimes arise without discernible cause. It is more prevalent in males than in females, occurring at any period of life. It is exceedingly rare as compared with hemorrhage into the cerebral meninges.

Pathology.—Hemorrhage into the spinal membranes is most frequently located in the cervical region. It is rarely very extensive, and compression of the cord is not pronounced. It may occur between the pia and the dura, or it may be subdural or subarachnoid. Subarachnoid hemorrhage often causes compression of the cord. Hemorrhages into the canal of the cord are very rarely noticed.

Symptoms.—Slight hemorrhages may take place without giving rise to symptoms, the onset, as a rule, being rather acute. Severe and sudden pain in the back, usually corresponding to the point at which the hemorrhage occurs, is the earliest symptom. This is accompanied by hyperesthesia and tingling in the extremities, particularly the arms. Muscular spasm is noticed early. Opisthotonos is not rare. There may even be general convulsive movements, due to irritation of the anterior nerve-roots. If the effusion of blood be sufficiently profuse to cause pressure upon the cord, anesthesia and paraplegia may result, the paralysis rarely being absolute. If the lesion occur at any place other than in the vicinity of the lumbar region, the knee-jerks are retained. Early in the course of the disease there may be retention of urine. Consciousness may be impaired early in the attack, but in the majority of cases the mind is unaffected.

Diagnosis.—The diagnosis depends upon the combination of pain with symptoms of nerve-root irritation without fever.

Prognosis.—Early in the course of the hemorrhage danger is most imminent in severe cases. It is graver in those cases which involve the cervical region, in which respiratory embarrassment is likely to arise. Quite a large number of cases recover. Paralytic and spastic symptoms may last for months, and even then may completely disappear.

Treatment.—Absolute rest is necessary, the patient lying upon the face or upon the side. Local abstraction of blood by cupping or scarification over the spine in the region of the pain has been advised, and ice-bags to the spine and morphin

hypodermically are useful. Violent purging should be avoided. If life is threatened, surgical interference should be tried. In intradural hemorrhages the membrane may be opened with success in some cases.

ANEMIA AND HYPEREMIA OF THE SPINAL CORD.

It is impossible to give a clear clinical description of anemia and hyperemia of the spinal cord. It has been experimentally proven that anemia of an area of the spinal cord abolishes functions, and, if long continued, may lead to necrosis of the nerve tissues; but this can result only from structural changes in blood-vessels. Mechanical congestion due to influence of gravitation may occur in weakened individuals and give rise to an aching sensation in the spine and legs when the body is in the recumbent posture. The vessels of the cord become dilated if there be prolonged or violent activity of the nerve elements, such as may take place in epilepsy, hydrophobia, or strychnin-poisoning, or after violent exercise.

PARALYSIS FROM LESSENED ATMOSPHERIC PRESSURE.

Definition.—A nervous disease characterized by paraplegia and nervous symptoms following exposure to increased atmospheric pressure.

Synonyms.—Caisson disease; divers' paralysis.

Etiology.—The production of this disease requires an increase of pressure of more than two atmospheres. The disease is due to the sudden reduction of atmospheric pressure which occurs on returning from a caisson to the outer air. If the lowering of pressure be very gradual, the symptoms rarely, if ever, arise. Alcoholism, chronic nephritis, myocarditis, and obesity are all predisposing factors. Novices are more likely to be affected than those who have been accustomed to work at gradually increasing depths. It is supposed that the risk of contracting the disease is increased when a person with an empty stomach enters a caisson.

Pathology.—It has been suggested that this disease is due to the liberation of gas (nitrogen) from the blood. Myelitis, hemorrhages, and congestion have been noticed at autopsies. The cause of the disease must still be regarded as being very obscure.

Symptoms.—The symptoms come on abruptly after a susceptible person leaves a caisson and returns to the surface. Occasionally the onset may be delayed for half an hour. Sometimes the symptoms consist of nausea, vomiting, tinnitus aurium, and severe pains in the joints. Collapse may come on rapidly, and death may result in a few hours. In milder cases the principal symptoms consist in tingling and pain in the extremities, which gradually disappear in a day or two. Under such circumstances no paralysis occurs. In the severer cases paralysis always takes place. It is rarely hemiplegic, mostly showing itself as a paraplegia. If the paralysis is complete, it may last for weeks, and then become permanent; if it is partial, recovery may occur in a few days. Anesthesia and implication of the sphincters take place in severe cases. Occasionally petechiæ have been observed.

Prognosis.—Less than 10% of the cases prove fatal. If death occurs, it takes place early. The cases that show cyanosis are serious even if no paralysis occurs.

Treatment.—The **prophylaxis** consists in compelling divers gradually to accustom themselves to increased pressure, limiting the exposure to a short time at first and increasing it by degrees. The diver should take food before entering the caisson.

For the attack, morphin cautiously given to relieve pain is valuable. Bandaging the limbs tightly has been recommended. The fluid extract of ergot in large doses is said to control the irritative symptoms.

HEMORRHAGE INTO THE SPINAL CORD.

Definition.—This term means that the hemorrhage does not occur between the membranes of the cord, but directly into its substance. It is a primary condition.

Synonym.—Spinal apoplexy.

Etiology.—Ninety per cent. of the cases are due to trauma, such as a fall upon the back, a blow, fractures and dislocations of the vertebræ, and violent muscular exertion. It may occur in the blood dyscrasias, especially in hemophilia. It may arise without apparent cause. The majority of cases have taken place between the ages of twenty and forty.

Pathology.—The hemorrhage may be very slight, but in rare instances it is more extensive. It is usually limited to the gray matter. The diameter of the cord about the site of the hemorrhage is somewhat enlarged.

Symptoms.—The onset is sudden; it may be associated with loss of consciousness, which is, however, of short duration. Most often sharp pain occurs in the affected region, with motor paralysis. Rapid wasting is common, and the paralysis takes the form of paraplegia. There may be slight anesthesia, but, as a rule, sensibility is not impaired. The temperature sense is not completely lost, but it may be greatly impaired.

Sequels.—Spastic paraplegia, contractures, and trophic changes of the skin, such as bed-sores, are common.

Prognosis.—If the symptoms are severe, death may take place in a few hours, but this does not often result unless the hemorrhage be very profuse. If wasting and paralysis remain for three months, very little improvement will take place, and the case may die from cystitis, bed-sores, or exhaustion. Complete recovery is very rare, as the contractures are almost always present in every case.

Treatment.—Absolute rest is necessary, and it is proper to have the patient lie upon the side or chest rather than upon the back. Ice-bags to the spine are useful. The fluid extract of ergot in large doses is said to have some power in controlling the hemorrhage. If pain should be associated, the administration of opium in full doses is better treatment. The bowels should be kept freely open by the use of salts, although continued purging should be avoided. In the early stages of the disease electricity is contraindicated.

ACUTE ANTERIOR POLIOMYELITIS.

Definition.—An acute disease of childhood, also occurring in adults, characterized by complete loss of power in one or more limbs, particularly in the legs, followed by atrophy of the muscles, and rarely by sensory disorders, coming on abruptly.

Synonyms.—Infantile spinal paralysis; acute atrophic paralysis; atrophic spinal paralysis.

Etiology.—The disease occurs in both sexes, most often, however, in young children. It has shown itself in children as young as five months of age. The disease occasionally shows an epidemic character. It is more common in the summer months. Exposure to cold, especially during perspiration, has been supposed to be a cause, as has also trauma. Of late the disease has been asserted to be of an infectious

nature, due to some micro-organism. This assertion has not been verified.

Pathology.—In the first stages of the disease there is hyperemia of the spinal membranes and of the gray matter. Upon microscopic examination numerous leukocytes and some red blood-cells are found in the affected area. There are also many small round cells, and the neuroglia reveals proliferative changes. Degenerative changes are noticed in the motor neurons. The cells are swollen and the outline is irregular and the protoplasm granular. The nucleus becomes obscure. The degeneration may continue until the vitality of the cells is entirely destroyed, the cells becoming very granular and evacuolated and the dendrites disappearing. Interstitial changes also accompany those just noted. From the atrophy of the cells the anterior horn undergoes shrinkage, and the fibers in the anterior nerve-root may also undergo degeneration and atrophic changes. From the extension of the atrophy, various groups of muscles become paralyzed. Sclerotic changes may also be noticed in the anterolateral column in the neighborhood of the lesion. It is believed by some that the disease is due to thrombosis of some of the spinal arteries or to hemorrhage into the anterior horn.

Symptoms.—As a rule, the disease begins suddenly, very much like an acute infectious disease, with convulsions, delirium, and fever. The temperature commonly rises to 102° F. or 103° F., accompanied by pain in the back and limbs, and occasionally by diarrhea. In rarer instances the disease is preceded by prodromes. In older children chill may occur at the onset. The temperature remains high for a few days, with slight morning remissions, and gradually falls to normal, the fever rarely lasting more than a week. Paralysis sets in rapidly in some instances; the child may go to bed apparently healthy and awaken the following morning with paralysis. Occasionally there is slight rigidity in the region of the spine. Bed-sores and trophic changes are exceedingly rare. Pain in the paralyzed limb is for the most part absent; at times slight pain is complained of in the region of the joints upon movement. Paralysis is usually more extensive early; later, improvement taking place. The muscles of the paralyzed limb soon undergo atrophy, so that there is decided change in the appearance of the limb within a month after the onset. The paralyzed limb is relaxed, never rigid, and the reaction of degeneration sets in rapidly. The galvanic response usually

remains for some time. The circulation in the affected area is markedly impaired; it becomes cold, flabby, and blue, rarely edematous. From disturbance of nutrition in the bone, shortening occurs, which remains permanently. Paralysis most frequently takes place in the legs, rarely is the face affected. Sensation is rarely interfered with. In some instances the entire muscular system appears to be attacked. Reflex action at the level of the lesion is affected in every case. Deformities of the joints are common sequels. The sphincters do not suffer in the general process.

Diagnosis.—Acute rheumatic fever may occasionally be confounded with the disease under discussion, but a careful examination will reveal local tenderness and pain in the joints, without atrophy, sweating, often some cardiac complication, all of which favor acute rheumatic fever.

Prognosis.—The disease is rarely fatal, but permanent paralysis in some part of the body frequently remains. Some improvement always takes place. If the muscles respond to the faradic current within three weeks after the onset of the disease, it is likely that the case will recover. The prognosis is better in the cases starting acutely with fever than in those beginning insidiously.

Treatment.—In the acute stage absolute rest in bed, with some mild counterirritant to the spine, is of use. Blistering is not desirable nor necessary. If the temperature is high, sponging with cold water and alcohol will be found effective. Ergot in small doses has been recommended. The iodids and salicylate of sodium are of some value. A mild laxative at the onset is useful. Late in the course of the attack, when paralysis begins to subside, strychnin in full doses, arsenic, cod-liver oil, or the hypophosphites may be beneficial. Care must be taken to preserve the nutrition of the muscles; this may be accomplished by hydrotherapy, massage, and electricity. In chronic cases orthopedic apparatus suited to the requirements of each individual case may be used.

ACUTE MYELITIS.

Definition.—Acute myelitis is an inflammation of the spinal cord, extending longitudinally, and most often involving the entire transverse area. The disease may be *diffuse*, *ascending*, or *descending*.

According to Leyden, the disease is subdivided from the

following points of view: (1) From the extent and region of the cord involved; (2) from the etiologic, and (3) from the clinical standpoint.

Etiology.—Exposure to variations of temperature, especially to cold, plays an important part in the causation of the disease; thus, certain occupations predispose, as those persons employed as engineers, cabmen, drivers, bakers, and so on. Gout, rheumatism, and certain metallic poisons, such as lead, mercury, and arsenic, have been recognized as etiologic factors. Alcohol may also be a cause. Syphilis usually gives rise to the chronic form. Occasionally acute myelitis may be secondary, resulting from extension, as in abscess, caries, or cancer of the spine. Traumatism is perhaps the commonest cause. The puerperal period and the existence of septic conditions have also been noted as causing the disease.

Pathology.—The cord may be swollen, the membranes congested, and on section the white and the gray matter are with difficulty differentiated. The consistency of the affected part is greatly reduced. The area may be reddened, owing to extravasation of blood (red softening). In some instances the entire cord may be affected (diffuse myelitis). If the condition persist and fatty degeneration ensue, and the hyperemia subside, the condition is then called yellow or white softening. Upon microscopic examination it will be found that the nerve-fibres are distorted and swollen. Numerous leukocytes and red blood-cells and fatty and granular cells are noted. The ganglion cells may also be irregular and swollen, their protoplasm showing degenerative changes. The nucleus may undergo division.

Symptoms.—The most important symptom is paralysis of motion, coming on rapidly, with complete loss of sensation below the site of the lesion, and paralysis of the sphincters (bladder and rectum). Bed-sores over the sacrum, the hips, and the heels, with some atrophy of the muscles, develop rapidly, usually within a week. Some rise in temperature is usually present. Convulsions are not infrequent. Most commonly the disease is situated in the lower dorsal region. After some weeks rigidity, with spasmodic jerkings upon peripheral irritation, occurs; more rarely, contractions take place involuntarily. Reactions of degeneration are not present. The reflexes are exaggerated when the lesion is above the lumbar region, and ankle clonus may be elicited. Loss of sensation is usually complete. The temperature sense is absent. Pain does not

occur. The skin soon becomes cold, and a clammy perspiration appears. Usually there is some edema. If the lesion be high up in the cervical region, there may be pupillary changes.

Complications and Sequels.—Cystitis is the most common complication. It is due to the retention of urine in the bladder. Extensive bed-sores have already been referred to. Secondary pulmonary and renal complications are common, and amyloid disease of the kidneys sometimes occurs.

Diagnosis.—The direct diagnosis depends on the sudden onset of the disease, with paraplegia, paralysis of the sphincters, loss of sensation, rapid trophic changes, and absence of pain in the muscles.

Prognosis.—Complete recovery is rare, more or less paraplegia usually remaining, as secondary changes are very likely to occur in the cord. The greater the amount of trophic change, the more unfavorable the prognosis. Prolonged high temperature is unfavorable. Cases due to syphilis are the most favorable.

Treatment.—Counterirritation by blisters or the application of cold are of use in the acute stage, but they are contraindicated after this stage. Precautions should be taken not to interfere with the nutrition of the skin, on account of the great liability to trophic changes. Absolute rest is important. Great care must be taken of the bladder; the catheter should be carefully sterilized. Electricity is beneficial late in the course of the disease, and at this time massage is also valuable. A warm climate favors improvement. Hydrotherapy has been highly recommended. General tonics are of use. The bowels may be moved by enemata. In the luetic cases antisyphilitic treatment is indicated.

DISSEMINATED MYELITIS.

Definition.—An acute disease of the spinal cord, often following the infectious fevers, such as smallpox and typhus, occasionally occurring independently of these affections.

Synonym.—Multiple myelitis.

Etiology.—The disease has most often been noted as occurring after the acute specific fevers, also as resulting from syphilis, alcohol, and exposure to cold. Occasionally the disease arises without assignable cause.

Pathology.—Sclerosis is found in many parts of the white substance of the cord, medulla, pons, and cerebrum, and some-

times in the gray substance. Red softening may also be noted. The nerves and the nerve-cells may undergo degeneration. Secondary degeneration is not often encountered.

Symptoms.—Sometimes the disease begins acutely, so that within a few days or a week there is ataxia and tremor of the extremities, usually in both the arms and legs; occasionally, however, this is limited to one side. There is a tremor of the head and of the tongue, and nystagmus is often present. Speech is altered; it is often slurring, sometimes explosive, and occasionally scarcely intelligible. The mental powers are interfered with to a decided extent. The face becomes expressionless, and often has a foolish appearance. The patient is emotional, easily excited, and has lost his power of self-control. There is much muscular weakness, which is rarely so pronounced as are the tremor and the incoordination. The deep reflexes are exaggerated, which condition later gives place to spastic contractions of the muscles. Sensory phenomena are not marked.

Complications.—Multiple sclerosis, optic neuritis, and some form of dementia are common complications. Death due to the disease itself is rare.

Diagnosis.—The direct diagnosis depends upon the suddenness of the onset, with the tremor and ataxia, and upon the mental phenomena and the course of the disease. Occasionally the disease resembles multiple sclerosis; however, in this the onset is more insidious, the mental symptoms are not so marked, and the entire course is more nearly chronic.

Prognosis.—Occasionally the disease may end in recovery after a few weeks. The prognosis, however, in general is unfavorable, as the disease is most likely to merge into multiple sclerosis. Fatal results occur from some intercurrent affections.

Treatment.—The treatment consists in rest in bed and the administration of iodid of potassium and ergot. Later, electricity, massage, and hydrotherapy are useful. Strychnin should be employed only when there is rigidity of the muscles.

CHRONIC MYELITIS.

Definition.—Chronic myelitis results from an acute or a subacute attack. The disease is essentially a sclerosis of the spinal cord. Rarely the disease may be primary.

Etiology.—The disease most frequently follows an acute attack. Under rare circumstances the chronic condition may come on from the effects of trauma or from hemorrhage, tumor, or caries in which destruction of the cord has occurred. Occasionally it is due to extension from surrounding tissues. In the rarest instances it is due to cold and syphilis.

Pathology.—Sclerosis is the most important lesion. The nerve-cells in the gray matter become atrophied and many disappear, and the nerve-fibers may also disappear and the blood-vessels may show thickening. Occasionally the membranes are sclerotic. The ascending and descending tracts are involved in the sclerotic process. Ascending degenerations are noticed in the posterior columns of the cord, extending as far as the medulla. The degeneration involves the columns of Goll. Gowers' column and the cerebellar tract may also disclose ascending degeneration. The lateral tracts of the cord show the descending degeneration.

Symptoms.—The symptoms are the same as in acute myelitis, differing, however, in the fact that they come on more insidiously, and are often less well defined. It may be some months before well-marked symptoms are noticeable. There may be numbness and tingling, with some degree of paresthesia; sensation, however, is rarely completely lost. The motor symptoms are gradual in their onset, and are rarely extreme, and the degree of trophic change depends upon the destruction of the cells in the anterior horns of the spinal cord. As the reflexes become exaggerated the spasticity rapidly increases. If degeneration in the posterior columns takes place, we find an increasing tendency to ataxia; this is most often marked in the upper extremities. Bed-sores appear late in the disease. The reaction of degeneration rarely occurs, although there is some decrease in the quantitative response.

Complications and Sequels.—Complications and sequels consist in the development of bed-sores, paralysis of the sphincters, and cystitis.

Diagnosis.—This is often difficult, and must at times be made by exclusion. It will depend upon the gradual onset of the symptoms just enumerated.

Prognosis.—The disease is incurable after the symptoms have once manifested themselves. The course of the disease extends over many years, death being due to exhaustion, cystitis, and bed-sores.

Treatment.—Rest in bed is most important, improvement

first occurring from this means alone. Hot baths, electricity, and massage are of value. Residence in a warm climate is often advantageous. Iodid of potassium has been recommended, but its utility is doubtful. Everything should be done to maintain the general health of the patient.

ACUTE ASCENDING PARALYSIS.

Definition.—A disease characterized by ascending paralysis, beginning in the lower extremities and extending upward, later involving the respirarory muscles and the heart.

Synonym.—Landry's paralysis.

Etiology.—Very little is known of the causation of this curious disease. From its acute onset and its course it has been supposed to be of an infectious nature. Occasionally the disease has followed exposure to cold and some one of the acute infections, such an enteric fever, variola, diphtheria, and so on. Alcohol and syphilis can not be put down as exciting factors. The disease occurs most frequently between the ages of twenty and fifty, the sexes being equally affected.

Pathology.—Little is known of the pathology. Various lesions, such as vascular disturbances, softening, extravasation of blood into the gray substance, have been observed.

Symptoms.—Prodromes commonly occur in this affection. Disturbances of sensation, paresthesia, and shooting pains are common. After a period of several days or weeks paralysis occurs in the lower extremities, which soon develops into complete paraplegia. The paralysis shows an ascending tendency—the legs first, next the body, then the muscles of the abdomen and back, without disturbances of respiration, and lastly there is paralysis of the arms. The reflexes disappear. Late in the course of the disease there are bulbar symptoms. There are disturbances of articulation and phonation and difficulty in chewing and swallowing. There are paralysis of the muscles of the eye, dilated pupils, increased pulse, and marked dyspnea. Death may take place in from two to three days, but ordinarily the disease lasts from one to two weeks. In some rare instances the paralysis shows a descending course instead of an ascending course. As a rule, the sphincters are normal.

Prognosis.—The prognosis is unfavorable.

Treatment.—Such stimulants as strychnin and digitalis are indicated for respiratory and cardiac stimulation. Massage and electricity are of use.

LOCOMOTOR ATAXIA.

Definition.—A very frequent disease of the spinal cord, characterized by progressive changes, with sharp, lancinating pains, incoordination, and paralysis.

The disease was systematically described by Romberg, in 1846.

Synonyms.—Tabes dorsalis; posterior sclerosis of the spinal cord.

Etiology.—The disease occurs most frequently in middle life, and particularly in the male sex. According to some observers, the history of syphilis is found in 90% of the cases. Sexual excess, trauma, and alcoholism have all been put down as etiologic factors.

Pathology.—The primary lesion is in the dorsal roots and in the tract of Lissauer. The extent of the sclerosis is in direct relationship to the duration of the disease. In the advanced stages it is wide-spread, so that the fasciculus of Goll reveals marked sclerosis in the later stages, while in the earlier stages this interstitial change is slight. The lesions in tabes are sclerosis of the posterior columns and the nerve-roots, degeneration of the peripheral and sensory nerves, and sometimes degenerative lesions of the cerebrum, optic nerves, and cerebellum. Even degenerative changes have been noticed in other sensory nerves. The cord presents marked shrinkage and thickening of the posterior columns, especially noted in the lower lumbar and dorsal regions, thinning of the posterior nerve-roots, and often the membranes covering the posterior portion of the cord are adherent.

Symptoms.—For convenience of description the disease has been divided into three more or less well-defined clinical stages: (1) The pre-ataxic stage; (2) the ataxic stage; and (3) the paralytic stage.

The Pre-ataxic Stage.—The symptoms which first call attention to the disease are the so-called "lightning pains." They are sudden in onset, sharp and shooting in character, and only momentary in duration. They frequently occur in paroxysms. Occasionally the pain is present in but one leg; at other times it is distributed, shifting its location rapidly. These pains arise most frequently in the lower extremities, occasionally in the upper, more rarely in the face. Dampness and cold aggravate the pain, so that the disease has commonly been mistaken for forms of so-called chronic rheumatism.

Slight cutaneous hyperesthesia often arises early in connection with this pain. Eye symptoms are very common in the first stage. There may be optic nerve atrophy, diplopia, strabismus, or ptosis due to some oculomotor palsy. The most important ocular phenomena relate to the pupil, which reacts to accommodation, but not to light. This is known as the "Argyll Robertson pupil." The pupil is most often small, and is called the "pin-point" pupil. Occasionally inequality in size occurs. Soon there may be slight difficulty in micturition. The early important symptom is the loss of the tendon reflex, the electric reaction of the extensor muscles remaining normal. The loss of the knee-jerk in tabes is known as *Westphal's sign.* In some rare instances the knee-jerk is preserved. The combination of the three symptoms—lightning pain, absence of tendon reflex, and eye phenomena—warrants a diagnosis of locomotor ataxia.

The second stage is noticed upon the occurrence of ataxic symptoms. By ataxia, or incoordination, is meant a want of harmony in muscular contraction as distinguished from a loss of power in the muscles. These symptoms first show themselves in walking or standing, beginning as a mere unsteadiness, which is increased by the patient closing his eyes. An early sign of which the patient complains, is that he can not walk in the dark. This incoordination steadily increases, so that occasionally when he attempts to turn, the feet are crossed. The ataxic gait soon manifests itself: the patient walks with his feet wide apart, soon depending upon a stick for support; he lifts the advancing foot high, throwing it outward with a jerky movement and bringing it down with a sudden stamp. When sitting, the patient often may not be able to describe a circle with his toe, and can not touch one knee with the opposite heel. When put in the erect posture with his feet close together and his eyes closed, he sways markedly; this being known as *Romberg's symptom.* Later in the disease he can not stand without support, and the incoordination may spread to the upper extremities. Soon it is impossible for the patient to execute the finer movements with the hands; thus, he can not pick up a pin from the table, he fails to button his coat, especially if his eyes are closed, he may not be able to touch the tip of his nose with his fingers, etc. By this time, as a rule, defects of sensation occur; there are tingling, a sensation of pricking by pins and needles, and numbness in the lower extremities, the patient often feeling

as if he were walking upon thick carpet or upon cotton. A peculiar sensation is felt around the waist, as if there were a tight girdle worn; this is known as the "*girdle pain.*" Anesthesia may now be present in various parts of the body; often there is analgesia, the patient not feeling the prick of a pin, the pain being markedly delayed. The symptoms of the first stage, as a rule, all continue throughout the second stage.

The third stage shows an aggravation of all the symptoms of the second stage, the patient often becoming quite helpless. There may be true paraplegia and paralysis of the sphincters, bladder, and rectum. The patient is now confined to bed or to a chair, becomes emaciated and feeble, bed-sores develop, cystitis is common, and intercurrent diseases may appear.

Description of Special Symptoms.—Motor Phenomena.—One of the earliest symptoms is often a disproportionate fatigue following slight exercise, accompanied by weakness in the legs.

Sensory Affections.—Hyperesthesia, analgesia, and paresthesia occur. Analgesia of the ulnar nerve particularly has been noted.

Visceral Crises.—These consist in a violent and sudden disturbance of the function of an organ, for which no sufficient cause can be given. The most frequent of these is the gastric crisis. This shows itself by a concentrated pain in the epigastrium, with severe and uncontrollable vomiting. The tongue is usually clean, the pulse frequent, and the temperature remains normal. These symptoms may be followed by severe nervous depression, and often by collapse. In a day or two the vomiting suddenly ceases, the patient being apparently restored to his normal health. After a variable period the attack recurs, being characterized in some cases by quite remarkable periodicity. As a rule, gastric crises occur early in the disease, and pass off as the disease advances. When the patient is questioned about these manifestations, he is apt to describe them as bilious attacks. Intestinal crises may be associated with the gastric crises, or occur independently. Similar conditions may arise in the region of the kidney, urethra, and bladder. The sexual function is often interfered with; it is sometimes exaggerated, and at other times entirely lost. Similar crises appear in the larynx, and are known as laryngeal crises, in which paroxysmal cough and dyspnea occur. Laryngeal crisis may be accompanied by coma or convulsions. Occasionally there is paralysis of the vocal cords.

Symptoms Referable to the Circulatory System.—Increased pulse frequency is a common symptom of tabes. Attacks which simulate angina pectoris occur, and are known as cardiac crises. Occasionally the symptoms of Graves' disease have been associated with tabes. Organic disease of the aortic valve is sometimes associated.

Trophic Lesions.—Charcot called attention to arthropathies, and the name "Charcot's joints" has been applied to the peculiar formations appearing at the joints in tabes. Any joint may suffer, but the large joints are more frequently attacked than the small ones, the knee-joints being the most commonly affected. The joint suddenly becomes swollen, and upon examination shows a large effusion. There is neither heat, tenderness, nor pain; occasionally new bone is developed and restrictions of movement occur. The joint rarely suppurates, but remains permanently enlarged. Degeneration of the arterial structures, bones, cartilages, and muscles is permanent. Great liability to fracture of the bones, especially the long bones of the extremities, has been noted in tabes. In some cases rupture of the tendo Achillis has been observed. Perforating ulcer of the foot is common, and is diagnostic. Anesthesia in the neighborhood of the ulcer is usual. Occasionally gangrene results. Deformity and loss of nails are common. Herpes sometimes occurs in the course of the lightning pains. Purpuric manifestations in the form of small subcutaneous ecchymoses are not infrequent.

Mental Affections.—In the majority of cases the mind is not affected. In rare instances mania may occur, and the form of general paralysis known as "tabes of the brain" has been noted.

Course of the Disease.—The course of the disease, as a rule, is slow, but progressive. The stages are measured by years rather than by months. Sometimes an apparent intermission takes place. It is very unusual for locomotor ataxia to run a rapid course, although cases that have lasted only a few weeks or a few months have been noted.

Diagnosis.—The diagnosis in a well-marked case is easy. Scarcely any other affection can be confounded with locomotor ataxia if the picture be typical. It must be differentiated from paraplegia, cerebellar disease, syringomyelia, and hysteria.

Prognosis.—The prognosis as to cure is extremely unfavorable. The duration of life is not always shortened by loco-

motor ataxia. Death may occur at any time from intercurrent affections. As a rule, optic nerve atrophy renders the prognosis of the spinal symptoms more favorable.

Treatment.—The treatment of locomotor ataxia is extremely unsatisfactory. It is impossible to prevent the development of the successive stages. Attention should be given to the general health and hygiene of the patient. For the relief of the lightning pains, antipyrin and drugs of this class are valuable; ten-grain doses repeated three or four times in as many hours usually afford prompt alleviation. For the crises morphin hypodermically is the only reliable agent. Hydrotherapy gives immediate relief in some cases. Electricity is also useful. Absolute rest in bed for a prolonged period of time has been urged by many authorities, especially during the time in which the lightning pains are severe. Good results may be obtained by the administration of the double chlorid of gold and sodium. In the majority of cases antisyphilitic treatment is valueless. When the use of the catheter becomes necessary, strict antisepsis must be insisted upon. The diet throughout the course of the disease should be liberal, attention being given to the condition of the bowels. The treatment by suspension is of value in some cases.

PRIMARY LATERAL SCLEROSIS.

Definition.—A chronic disease of the spinal cord, due to sclerosis of the descending fibers of the crossed pyramidal tracts, with marked symptoms referable to the nervous system.

Synonyms.—Spastic paraplegia; spastic spinal paralysis; Erb's palsy.

Etiology.—The disease is most common in adults between the ages of twenty and forty, both sexes being equally affected. Infective processes, sexual excess, syphilis, and lead-poisoning have been designated as etiologic factors. It is more common in neurotic families.

Pathology.—In this disease the crossed pyramidal tracts reveal sclerotic changes. The multipolar cells in the anterior cornua may be involved in the sclerotic process. Disseminated sclerosis may follow lateral sclerosis.

Symptoms.—The disease begins insidiously with symptoms of fatigue upon slight exertion, and there is some alteration in the gait. The knee-jerks are markedly increased. These symptoms progress in severity until the fatigue is so great

that the patient walks with much difficulty. When he attempts to walk, the muscles are drawn into a state of extreme tension. Clonic spasms are apt to occur in the feet and legs, especially when the patient is in the recumbent posture. Besides the increased knee-jerk, ankle clonus is present. The disease may extend high up in the cord and affect the upper extremity. The skin reflexes are also increased. Other functions of the nervous system are not impaired. The movement of the patient becomes difficult on account of the spasmodic condition of the limbs. Walking is performed slowly and with effort. The feet can not be lifted clear of the ground, and so the front part of the foot is dragged with each step. This often shows itself in examining the shoe. In the later stages of the disease it is impossible for the patient to walk at all.

Prognosis.—The prognosis is unfavorable. The condition is incurable.

Treatment.—The treatment is the same as in other forms of cord disease. Warm baths have been highly recommended to relieve the spastic condition of the muscles. A good general diet is of use. General tonics such as cod-liver oil, hypophosphites, and arsenic may be of value. If syphilis be suspected as a causative factor, mercury and iodid of potassium should be tried.

POSTEROLATERAL SPINAL SCLEROSIS.

Definition.—A sclerosis of the posterior and lateral columns of the spinal cord, with symptoms of lateral sclerosis, mostly preceded and generally accompanied by those of sclerosis of the posterior columns of the cord.

Synonyms.—Progressive spastic ataxia ; ataxic paraplegia.

Etiology.—The disease occurs most frequently between the ages of thirty and fifty, and is more common in men than in women. Little is known of the etiology.

Pathology.—Very often this disease represents an extension of the sclerotic process of tabes into the lateral columns, or it may be secondary to myelitis.

Symptoms.—The symptoms are those of spastic paraplegia combined with incoordination. The onset is insidious. All the symptoms of spastic paraplegia occur, but as the spastic phenomena become marked in the lower extremities, the signs of incoordination also appear. The gait has the

combined qualities of locomotor ataxia and lateral sclerosis. The reflexes are most often increased. Ankle clonus may be present. Lightning pains are, as a rule, absent. Anesthesia does not occur. The Argyll Robertson pupil is rarely present. Later in the course of the disease the spastic phenomena become more prominent, so that the case is often mistaken for one of advanced lateral sclerosis. If the ataxic phenomena are more prominent, the knee-jerks may disappear and the case more closely resemble tabes. The symptoms, after they have involved the lower extremities, gradually extend to the upper extremities.

Prognosis.—The course of the malady is progressive, although slow.

Treatment.—The treatment is the same as that described for sclerosis of the cord in general.

PUTNAM AND DANA'S COMBINED SCLEROSIS OF THE LATERAL AND POSTERIOR COLUMNS.

This disease consists in a subacute sclerosis of the lateral and posterior columns of the cord, in which softening finally occurs at certain levels. It is characterized by symptoms of spastic paraplegia, but develops with more rapidity, death taking place much more speedily than in other forms of sclerosis.

Etiology.—The disease is said to be due to chronic lead- or arsenic-poisoning. It is more common in women, and may be associated with pernicious anemia.

Pathology.—The lesions are those of the rapidly progressing sclerosis of the lateral and posterior columns, accompanied by an acute inflammation of the substance of the cord.

Symptoms.—The disease begins with numbness and tingling in the extremities, particularly in the feet, with great emaciation and anemia; obstinate diarrhea is often present. The first symptom referable to the nervous system may be paraplegia. The knee-jerks are exaggerated and ankle clonus is present. The lower extremity is much more frequently affected than the upper. Girdle and lightning pains are rarely present. There are no eye symptoms. The disease proves fatal in two years from its commencement.

Prognosis.—The prognosis is absolutely unfavorable.

Treatment.—The treatment should be palliative.

PELLAGRA.

This is a rare disease in this part of the world, but occurs in parts of Italy, France, and Spain. In this disease the anatomic changes show a degenerative process in the posterior and lateral columns of the spinal cord, associated with atrophy of the large cells in the anterior horns. There is also a sclerosis of the pia mater, and occasionally there is a formation of bony plates in the arachnoid. Pellagra is said to be due to poisoning from diseased maize. The symptoms are those of ataxic paraplegia associated with marked wasting.

HEREDITARY ATAXIC PARAPLEGIA.

Definition.—A rare disease of the spinal cord occurring in families, especially in young individuals, characterized by paraplegia and accompanied by changes in the lateral and posterior columns of the spinal cord.

Synonym.—Friedreich's ataxia.

Etiology.—The occurrence of two or more cases in one family is characteristic. The consanguinity of parents has been traced in a certain proportion of cases as a cause of the disease. The sexes are equally affected. The disease may affect only one sex in a family to the exclusion of the other. The acute infectious diseases have been given as etiologic factors. Ten cases have been known to occur in the same family.

Pathology.—Little is known of the pathology in this condition. In many cases reduction in the diameter of the spinal cord is noted, probably due to hypoplasia or atrophy, the latter being the result of fibrous connective-tissue contraction, for in nearly all cases fibroid changes are noted, particularly of the posterior columns. The direct cerebellar tract or direct pyramidal tract, the lateral pyramidal, and the tract of Lissauer are sometimes involved. The cells in the anterior and posterior horns of the columns, as well as the cells of Clarke, may be atrophied.

Symptoms.—The first symptoms occur at any time from the second to the twenty-fourth years. In the majority of cases they appear between the sixth and the fifteenth years. Several members of the same family may be affected. Rarely isolated cases occur. The symptoms are those of spastic paraplegia and locomotor ataxia, with marked wasting. Irregular move-

ments may take place in the limbs, even when the patient is at rest. Speech becomes impaired early; it is thick and monotonous, and sounds as though the patient had a foreign body in his mouth. Occasionally there are jerky movements in the tongue. The face is expressionless, but the intellect is unaffected. Nystagmus is a common symptom. The pupillary reactions are normal. The deep reflexes are absent from the beginning of the attack. Sensory symptoms are rare. A peculiar deformity of the foot occurs, known as the "pes cavus," consisting of a peculiar stumpy and jerky appearance from before backward, in which the arch is markedly exaggerated, the toes being extended; the great toe is unusually prominent. A similar deformity sometimes occurs in the hand, known as "manus cava." Death may result from malnutrition or from intercurrent diseases.

Prognosis.—The prognosis is unfavorable.

Treatment.—The treatment is the same as in other forms of sclerosis.

AMYOTROPHIC LATERAL SCLEROSIS.

Definition.—The disease is characterized by a chronic progressive muscular atrophy, usually at first limited to one part of the body, most often the upper extremity, gradually spreading and involving other parts.

Synonyms.—Spinal muscular atrophy; wasting palsy.

Etiology.—It attacks the patient between the ages of twenty-five and fifty, usually when over thirty. Women are more often affected than men. Scarcity of food, exposure to cold, and injuries to the back have been given as etiologic factors.

Pathology.—The changes are similar to those found in infantile spinal paralysis, atrophy of the ganglion cells in the anterior horns and sclerosis of the lateral columns are found. It is a degenerative process. The dorsal and lumbar regions, but especially the cervical, are affected. The anterior root-fibers are atrophied and the anterior commissure is degenerated. Degenerated fibers are also found in the peripheral nerves with atrophy of the muscles.

Symptoms.—Gradual wasting of the muscles is the most striking symptom of this disease. In 90% of the cases the muscular atrophy begins in the arms, and particularly in the hands, or in the muscles of the shoulder. When the atrophy

begins in the muscles of the hand, the thenar and hypothenar eminences (thumb and little finger) are first affected, which is a striking feature of this disease. Usually the atrophy is unilateral, especially at the beginning of the malady, and it may be a year or more until the other side becomes affected. The atrophy soon spreads to the other muscles, such as the muscles of the back and neck. If the respiratory muscles atrophy, life is shortened. In rare instances the atrophy may begin in the lower extremity. Loss of power is always marked, and is proportionate to the degree of wasting. Fibrillary contraction commonly occurs in the atrophied muscles. This is quite characteristic. When the wasting in the muscles is rapid, there may be partial or complete reactions of degeneration. The tendon reflexes are exaggerated; if wasting occurs in the lower extremity, the knee-jerk is lost. Occasionally there are rigidity and a spastic condition of the muscles. The affected muscles are flaccid when not in a spastic condition. All grades of atrophy may occur in different cases. The sphincters, as a rule, are unaffected. The sexual power is frequently lost.

Prognosis.—The prognosis both as to life and improvement of the wasted muscles is grave. It may prove fatal in less than a year from the onset, or it may last for fifteen or twenty years. The ordinary duration is from three to five years. If the disease shows a rapid onset, the course is liable to be short. The chief danger to life is due to involvement of the muscles of respiration and to bulbar paralysis.

Treatment.—Gowers favors the injection of nitrate of strychnin into the muscles, beginning with the minimum and rapidly increasing to the maximum dose. Fowler's solution should be given by the mouth. Electricity ought not to be employed. Massage and passive movements are said to be of service. The patient should be placed amid the best hygienic surroundings.

HEREDITARY OR INFANTILE FORMS OF PROGRESSIVE ATROPHY OF SPINAL ORIGIN.

Werding and Hoffmann have reported cases in which several children in the same family were affected during the first year of life by weakness and symmetric wasting in the muscles of the legs and back, extending upward. The paralysis was of the flaccid type, and accompanied by reactions of

degeneration, absence of knee-jerks, and incomplete loss of the skin reflexes. Lordosis of the lumbar spine was present, and some bulbar symptoms were noted. There was no pain or tenderness. There were no alterations of sensibility. The mental power was well preserved and the sphincters were normal, death taking place in a few years from involvement of the muscles of respiration. The following pathologic lesions were noted: A degenerative atrophy of the ganglion cells of the anterior horns; marked degeneration of the anterior nerve-roots; degeneration of the mixed nerves; and secondary atrophy of the muscle-fibers, with an increase of the nuclei of the sarcolemma. This has been regarded as a separate type.

PROGRESSIVE MUSCULAR ATROPHY OF THE HEUBNER-STRÜMPELL VARIETY.

The same symptoms occur in this variety as in the form already described, except fibrillary contraction of the muscles and the appearance of the reactions of degeneration. Occasionally even some of the muscle-fibers show hypertrophy. It has been asserted that in these cases the changes in the spinal cord are secondary to those in the muscular system. It is still a question whether this disease is a nosologic entity.

DISSEMINATED SCLEROSIS.

Definition.—A sclerosis of parts of the brain and spinal cord, with characteristic phenomena.

Synonyms.—Multiple sclerosis; insular sclerosis; cerebrospinal sclerosis.

Etiology.—The disease occurs in early adult life. It is rare after the age of forty. It probably appears more often in females than in males, and it has been supposed that the disease is more likely to arise in females with a neurotic tendency. It may follow chronic lead-poisoning and the excessive use of alcohol. The acute infectious diseases also appear to have some causative relation. Cold, exposure, trauma, and fatigue have been found to precede many cases.

Pathology.—Sclerotic areas are found in various parts of the nervous system, particularly the central nervous tissue, sometimes the cranial and other nerves. The sclerotic tissues

are represented by reddish-gray patches or very firm areas of a lighter color. The cord, the pons, the crus, the cerebrum, and the cerebellum have all been found involved. Upon contraction of the fibrous connective tissue atrophy of the nerve-tissues ensues. The blood-vessels frequently reveal sclerotic changes.

Symptoms.—Three distinct types of this disease have been described: The cerebrospinal, the cerebral, and the spinal. In the majority of cases, however, the disease involves both the brain and the spinal cord; and hence the predominant type is the cerebrospinal. The first symptoms may appear after some mental or physical strain, the patient behaving as if affected by hysteria. There may be temporary aphonia, from which there may be speedy recovery, or numbness may occur in some part of the body. There may also be a sensation of deadness and of coldness, a feeling as of the pricking of pins and needles, or there is great loss of power in some part of the body, even with convulsions. These symptoms may disappear for a time and reappear with increased severity. When the disease has been fully established, periods of remission may occur. A common type of the disease as described by Charcot shows the following symptoms: The patient develops spastic paraplegia, with exaggerated reflexes, with ankle clonus, and limited movements. An irregular jerky tension tremor occurs, nystagmus being a prominent symptom. Diplopia and paralysis of the ocular muscles are common. Scanning speech is prominent. There are paresthesia, tinnitus aurium, and vertigo. The mental faculties are blunted, and in some few cases apoplectiform and epileptiform convulsions may appear. Trophic changes arise late in the disease, the sphincters, however, remaining normal. Another variety of the disease is one in which the symptoms are more closely referable to the spinal cord, in which the cerebral manifestations, such as the vertigo and apoplectic attacks, the ocular symptoms, scanning speech, tension tremor, and so on, do not occur. Rarely are the knee-jerks absent.

Prognosis.—The prognosis is unfavorable. The disease may last for a long time. The cases in which the spinal symptoms alone are present may last for twenty years or longer.

Treatment.—The treatment is the same as in other forms of sclerosis. The salts of gold, silver, and arsenic have been recommended.

TUMORS OF THE SPINAL CORD.

Tumors may arise from the membrane and from the cord substance. The following tumors have been found arising from the membranes: Lipomata, myxomata, enchondromata, fibrolipomata, sarcomata, and carcinomata. Sarcomata, as a rule, are primary, but carcinomata are always secondary. The tumors arising from the cord substance are, as a rule, primary, the following having been noted: Myxomata, fibromata, psammomata, sarcomata, fibrosarcomata, angiosarcomata, neuromata, and lipomata. Echinococcus cysts have also been found. As a rule, but one tumor has been found. Sarcoma and neuroma have occasionally been found multiple. Gummata have been noted. It is supposed that trauma may have some influence in the production of new growths.

Symptoms.—The symptoms are those of compression and local irritation. At first the pain is local, associated with stiffness; later, atrophic paralysis associated with anesthesia and hyperesthesia occurs. Spasms with exaggerated reflexes and disturbance of sensibility are common. Paraplegia with paralysis of the sphincters is a late symptom.

Prognosis.—The prognosis is unfavorable, except in the cases of gummata.

Treatment.—If the disease be of syphilitic origin, iodid of potassium and mercury are of use. Surgical interference is often indicated. For the pain opium and counterirritation are necessary.

SYRINGOMYELIA.

Definition.—Syringomyelia is characterized by the formation of a cavity in the substance of the spinal cord owing to the breaking-down of certain nerve-structures.

Synonym.—Gliosis spinalis.

Etiology.—The disease may begin after severe trauma to the spine. Some cases have been known to follow the infectious fevers, especially enteric fever, most cases, however, occurring between the twentieth and thirtieth years of life, and being slightly more common in males than in females. Nothing is definitely known of the etiology.

Pathology.—This disease is regarded as a gliosis with degeneration of the central portion of the cord and hemorrhages, so that a cavity is formed in the central portion of the cord. It is to be distinguished from hydromyelia, which simply refers to

a distention of the central portion of the canal. The cavity in the cord in syringomyelia may involve the entire length, but more frequently it is localized to certain areas. The cavity formation frequently involves the upper part of the cord, the cervical and thoracic regions. A brownish gelatinous fluid is sometimes contained in the central portion of the cord.

Symptoms.—The recognition of the disease depends upon the association of three important symptom groups: (1) Loss of the sensations of pain and temperature in any part of the body, the tactile sense being preserved in the same area; (2) trophic changes in the skin, muscles, bones, or joints; (3) progressive muscular atrophy, with paralysis. With these symptoms there may be associated spastic paraplegia or the symptoms of a transverse myelitis or the symptoms relating to involvement of the lateral, posterior, or all the columns of the spinal cord. The prominence of the symptoms will depend upon the distribution of the lesion. The course of the disease is chronic, the symptoms appearing gradually, and remaining stationary for years, death being due to some intercurrent affection, although in rare instances death has occurred from the rupture of the cavity. In a large number of cases there is marked curvature of the spine, either laterally or forward or occasionally even backward. The disease formerly called Morvan's disease is now known to be a variety of syringomyelia, as in all cases examined after death the cavity has been found in the spinal cord. The reflexes vary; they may be either lost or exaggerated, depending upon the situation of the lesion; and if the disease extend to the medulla, implication of the cranial nerves occurs.

Prognosis.—The prognosis is unfavorable, the disease lasting for many years.

Treatment.—The treatment is symptomatic. If the paralysis occurs, it should be treated as an anterior poliomyelitis. If there are marked trophic changes, they should be treated by rest, by mechanical appliances, and by surgical means. The sensory loss can not be remedied. Tonics may be necessary.

DISEASES OF THE MEDULLA AND PONS.

PROGRESSIVE BULBAR PARALYSIS.

Definition.—A disease of middle age, characterized by impairment of speech, phonation, mastication, and deglutition, which is both gradual and progressive, with increasing muscular atrophy.

Synonyms.—Labioglossolaryngeal paralysis ; chronic bulbar paralysis ; Duchenne's disease.

Etiology.—This disease rarely occurs before the forty-fifth year of life, and is more common in the male sex. It is supposed that lead-poisoning and diphtheria may give rise to it. Exposure to cold, trauma, and syphilis have been named as etiologic factors.

Pathology.—The muscular atrophy is limited to the lips, tongue, palate, and muscles of the larynx, and if spinal atrophy be associated, the muscles of the neck, shoulders, or arms are also involved in the process. The tip of the tongue frequently reveals very marked atrophy. The roots of the hypoglossal, glossopharyngeal, vagus, facial, the motor nucleus of the fifth, and spinal accessory may show distinct atrophy. They are of a grayish-red color and very much thinned. Changes are also encountered in the medulla oblongata. The ganglion cells in the motor nuclei are atrophied. The most marked atrophy is found in the nuclei of the hypoglossal, and is less marked in the nuclei of the vagus and spinal accessory. Changes have also been noted in the cord.

Symptoms.—The first signs of the disease are some involvement of the tongue, so that speech becomes indistinct, especially in the use of the linguals, such as "l" and "n." The lips soon become weak, and the patient has difficulty in pronouncing the labials. Whistling is impossible. Next the muscles of the pharynx and palate suffer. Saliva dribbles from the mouth ; the face becomes motionless and without expression. Speech becomes impossible. There is total inability to swallow. Some atrophy of the muscles of the face also takes place. Sensory phenomena are not present ; however, the throat reflexes are early lost, so that food finds its way into the larynx. There are slight, if any, changes in the electric reactions. The mind is not impaired ; the patient, however, is apt to be emotionless. Progressive muscular atrophy is a

common association. The advance of the disease is progressive, although there may be periods of intermission lasting some weeks or months. The duration of the disease varies from six months to nine or ten years, the majority of cases lasting from two to four years.

Prognosis.—The prognosis is unfavorable, especially so if the disease show itself early in life. The principal causes of death are inanition, bronchopneumonia (deglutition pneumonia), failure of respiration, and intercurrent diseases (croupous pneumonia).

Treatment.—The general health of the patient must be maintained. Tonics, such as arsenic, iron, and strychnin, are useful. Great care must be taken to prevent food from finding its way into the larynx. In the later stages of the disease morphin hypodermically should be used to make the patient comfortable.

HEMORRHAGE INTO THE MEDULLA AND PONS.

Hemorrhage into the medulla and pons frequently occurs by extension or from the rupture of a bulbar blood-vessel.

Etiology.—Hemorrhage in this locality is due to the same causes that give rise to hemorrhage in general. Hemorrhage into the medulla is rare, and into the pons is also very rare as compared with hemorrhage into the internal capsule.

Pathology.—The causes of this condition are similar to those of cerebral hemorrhage, and it may result from trauma, miliary aneurysms, meningitis, syringomyelia, or tumors.

Symptoms.—As a rule, death occurs very rapidly—in from a few seconds to a few minutes—from interference with the cardiac and respiratory nuclei. There is loss of consciousness, most often without convulsions. In the rarest instances recovery may follow with symptoms of bulbar paralysis. When convulsions occur, they commonly begin at the onset of the affection. They are usually general and epileptiform in character, but the legs alone may be affected. Paralysis is often bilateral; rarely hemiplegia occurs. The pupils are "pin-point," but in rare instances may be dilated. Vomiting is common, the temperature is high,—from 105° F. to 106° F.,—and the respiration is of the Cheyne-Stokes type.

Prognosis.—Recoveries are extremely rare. At least four-fifths of all cases are fatal. It should always be borne in

mind that if recovery takes place, secondary attacks are most likely to occur.

Treatment.—The treatment is that of cerebral hemorrhage. (See p. 699.)

DISEASES OF THE BRAIN.

EXTERNAL PACHYMENINGITIS.

Etiology.—This is an exceedingly rare disease, occurring especially as a so-called idiopathic affection. It usually follows trauma to the skull, diseases of the bones, or inflammatory conditions of the muscles or other parts covering the bone. Concussion of the head may also give rise to it. As a rule, there is an exudation of blood from vessels that are engorged between the dura mater and the bone.

Symptoms.—The symptoms are those of cerebral compression. Only from these symptoms is it possible to arrive at a diagnosis. The condition, as a rule, belongs to the domain of surgery. Occasionally pus formation takes place, and in this way the pia-arachnoid may become affected, and leptomeningitis develop.

Treatment.—If a purulent inflammation can be diagnosticated, the trephine must be used as early as possible. Mercury and iodid of potassium have been advised.

INTERNAL PACHYMENINGITIS.

Synonyms.—Hemorrhagic pachymeningitis; hematoma of the dura mater.

This condition of the meninges consists of newly developed fibrous connective tissue between the dura and the internal membranes. Numerous hemorrhages into this new-formed membrane are found, some of which are very minute; again, very large extravasations of blood are found, hence the name "hematoma of the dura mater." The lesion at first always attacks the dura at the point where the hemorrhage arises; it is associated with diseases of the blood-vessel walls, such as atrophic changes of the vessels, with chronic alcoholism, senile dementia, and dementia paralytica. The lesion is usually bilateral, and very often situated in association with the parietal bones.

The condition has been observed as occurring with senile

changes, also with leukemia, pernicious anemia, and diseases of the heart.

The symptoms are by no means characteristic. There may be headache, dizziness, and sometimes convulsive movements. If the hemorrhage be large, symptoms of pressure arise.

Treatment.—The treatment is not at all satisfactory. General tonics are of use. If the hemorrhage be extensive, surgical treatment may serve to benefit the patient.

ACUTE LEPTOMENINGITIS.

Synonym.—Inflammation of the pia-arachnoid.

Etiology.—This condition most frequently arises from extension of adjacent points of infection to the pia and arachnoid. Caries of the bones of the skull is a common cause, and thus micro-organisms gain entrance. The disease most frequently arises from the temporal bone, especially from middle ear disease, and often as a result of an otitis media. Infection may occur from diseases of the sinuses, such as the frontal, the ethmoidal, and the sphenoidal. Trauma is a common cause. Acute leptomeningitis may develop during the course of erysipelas, pneumonia, septicemia, enteric fever, scarlet fever, measles, ulcerative endocarditis, acute rheumatic fever, and sometimes in some of the chronic diseases, such as gout, arteriosclerosis, and renal disease.

Pathology.—The lesion may be diffuse or localized to certain areas. Unilateral disease often is due to extension from middle ear disease. The exudate varies from a fibrinous to a purulent one, and in some cases may be hemorrhagic. Various micro-organisms have been associated with this disease, particularly the pneumococcus, the streptococcus, the staphylococcus, the gonococcus, the bacillus typhosus, and the bacillus coli communis.

Symptoms.—As a rule, headache is a prominent symptom in this affection; this is usually continuous and severe. Generally vertigo is present. Delirium soon manifests itself, and may alternate with coma. There is photophobia and great aversion to the slightest sounds, even while the patient is sleeping or partly somnolent. There is general cutaneous and muscular hyperesthesia. Vomiting of the cerebral type is common. If pus-formation takes place, chills occur followed by fever of a septic type. The temperature may be normal or, under exceedingly rare circumstances,

subnormal. In young children hyperpyrexia is common and the pulse-rate is very rapid, but there is no proportion between pulse and temperature. Occasionally there is bradycardia. Convulsions are common, and they may affect the muscles of the face or of the extremities. Painful contractions of the muscles of the back of the neck are frequent in basilar meningitis. The belly wall is often rigid and the abdomen may be retracted. If the membranes at the base of the brain be particularly affected, the nerves of special sense may become involved, the most common being an involvement of the eye, producing strabismus, ptosis, and a variation in the size of the pupils. If the seventh nerve be affected, facial paralysis appears. Optic neuritis has been noted in some cases, and may occur even without pressure upon the optic nerve. Hemiplegia and monoplegia are common. In children the respiration is quickened, sighing, and irregular, Cheyne-Stokes respiration being not uncommon. Constipation is the rule. Herpes is encountered in this form of meningitis.

Prognosis.—The prognosis is unfavorable.

Treatment.—Absolute rest and perfect quiet are essential. Local blood-letting in robust individuals is useful, especially in the early stages of the disease. Ice-bags should be freely applied to the head. Laxatives are of benefit, calomel and the salines being the most useful. For the hyperpyrexia cold baths are indicated. The bromids are of great value. Mercury and iodid of potassium are highly recommended in the treatment, and should be given in full doses. The food should be nutritious and consist largely of liquids. If vomiting persist, rectal alimentation becomes necessary. Administration of the perchlorid of iron is to be recommended.

CHRONIC LEPTOMENINGITIS.

Etiology and Pathology.—This disease may arise from syphilis, from the use of alcohol, and from some of the acute varieties of meningitis, which, having run a prolonged course, may have become chronic. The lesions consist of thickening and gluing together of the membranes. The extent of the process is usually circumscribed.

The symptoms are not distinctive. There may be headache, dizziness, and vomiting. At times some of the cranial nerves may be involved.

The treatment consists in the administration of opium, the iodids, and mercury, but little is to be hoped for, even from these measures.

CEREBRAL HEMORRHAGE.

Synonym.—Cerebral apoplexy.

Etiology.—Heredity is of importance in this affection, the apoplectic constitution being present in many individuals. Diseases of the blood-vessel walls are the most important factors in the etiology, many diseases giving rise to these changes. (See Diseases of the Blood-vessels.) It may follow some of the infectious fevers, and may also exist in diseases producing disturbances of the blood. Embolism is an important factor. Hypertrophy of the heart, exertion, or excitement frequently cause rupture of the diseased blood-vessels. It occurs at all ages, but is more common after middle life, males being more often affected than females in the proportion of four to one.

Pathology.—The lesions found are atheromatous blood-vessels and miliary aneurysms that have ruptured. Disease of the blood-vessel walls may arise from an infected embolus blocking up the vessel, the wall becoming diseased and an aneurysm developing, which subsequently ruptures. The lenticulostriated arteries, branches of the middle cerebral, are the vessels that most frequently give rise to cerebral hemorrhage. Lesions of other parts of the body, such as chronic interstitial nephritis, hypertrophy of the heart, and arteriosclerosis, are often associated with this condition. If the hemorrhage be slowly produced, clotting or coagulation follows, only a small area being involved; subsequently the clot may undergo softening. The surrounding brain-substance may become somewhat discolored, and in some instances a yellowish-brown fluid may remain, which is surrounded by the cyst-wall; this is called an apoplectic cyst. Very frequently the brain-substance surrounding the area of hemorrhage undergoes degeneration and softening. If the hemorrhage be extensive, marked destruction of the brain tissues will result. If the hemorrhage be very minute, absorption takes place in some instances, leaving only a small yellow patch. The most frequent situation of cerebral hemorrhage is in the corpus striatum, near the lenticular nucleus, more often involving the left than the right side in this situation. The pons Varolii is next in frequency in the order of involvement, the white matter of the frontal region being next. The optic thalamus may also be

involved. Hemorrhage may take place into the lateral ventricles; it is rarely primary. Meningeal hemorrhage or hemorrhage into the arachnoid space is sometimes encountered. Extradural hemorrhage is most often of traumatic origin, and this is more frequently due to the rupture of the middle meningeal artery than to lesions of other vessels. Subdural hemorrhage due to rupture of the middle cerebral may also arise from tumors, such as sarcomata. Secondary degeneration of the pyramidal fibers of the medulla and cord ensues if the hemorrhage does not terminate fatally. The degenerated nerves are finally replaced by fibrous connective tissue. In some instances the hemorrhage is so profuse that it may involve the whole of the cerebrum. If the hemorrhage be small and be absorbed, in some instances a cicatrix remains, which is altered by blood-pigments, and is called an *apoplectic cicatrix*.

Symptoms.—As a rule, prodromes do not occur; but when present, they consist of vertigo, some mental irritability, numbness or tingling in the extremities, and headache. Occasionally there may be sudden loss of speech. In the majority of instances the onset of the attack is sudden, with or without loss of consciousness. The suddenness with which the attack comes on has given rise to the term "apoplectic stroke" (Schlaganfall). There may be sudden sharp pain in the head at the onset; in other cases the beginning of the attack is painless. In the majority of instances when the loss of blood has been profuse, oncoming coma rapidly follows. It occasionally happens that the patient partially or completely recovers consciousness, or the unconsciousness from the initial attack may become more intense, this condition lasting for hours or days before death ensues. This has been called *ingravescent apoplexy*. The skin soon is bathed in perspiration. The face and neck are turgid and the countenance is cyanosed; on the other hand, in the severest cases there may be pallor. The breathing is stertorous. As a consequence of fluid in the trachea, loud rales are heard. The cough reflexes are diminished, as are also the other reflexes. The cheeks are flaccid, and are drawn in and puffed out with each respiratory act. The respirations are irregular, deep, and slow; Cheyne-Stokes respiration occurring in fatal cases. The pulse varies, commonly it is slow and of good volume; rarely it may be rapid, and an irregular pulse is of unfavorable prognostic import. Vomiting is common early in the attack. Often the head is turned to one side, accompanied

by a conjugate deviation of the eyes in the same direction, the patient looking toward the lesion. This symptom is not of long duration and disappears after two or three days. During a convulsion the deviation may be in an opposite direction; in the case of hemorrhage into the pons the eyes look away from the lesion. The pupils may be either dilated or contracted; commonly they are unequal. The temperature varies in individual cases; in large hemorrhages that rapidly prove fatal subnormal temperature is common early in the attack; thus, temperatures of 94° F. have been recorded. As a rule, reaction occurs after the fall, which may be either gradual or rapid. In the milder cases the temperature rises to a little above normal, and in the severer cases it may reach 108° F. or higher. Commonly there is a difference between the two sides of the body as regards temperature. The sphincters of the bladder and rectum are relaxed. The urine is often copious in amount, and may contain albumin and sugar. One of the most important symptoms of apoplexy is hemiplegia. During the deep coma it is often difficult to tell which side is paralyzed, but as the coma subsides movements are often noticed upon the unaffected side.

Crossed Hemiplegia.—Crossed hemiplegia may result from hemorrhages into the crus, the pons, or the medulla. There is a loss of function of one side of the body, and also a loss of function in some cranial nerves of the opposite side.

Symptoms of Hemorrhage into the Crus.—The symptoms are paralysis of the oculomotor nerve corresponding to the side upon which the hemorrhage occurs, and paralysis of the arm, the face, and the leg of the opposite side. If the geniculate body which is in relationship to the crus be compressed, hemianopsia arises.

Symptoms of Hemorrhage into the Pons Varolii.—The symptoms of hemorrhage in this locality are quite characteristic. As a rule, the coma is marked. General convulsions occur, which are rarely unilateral; they may, however, affect only the lower extremity. As a rule, the pupils are contracted. The temperature falls a degree or so early in the attack, but in a very few hours rises rapidly, and hyperpyrexia is noted. With involvement of the seventh nerve there is paralysis of the face on the side of the lesion, hemiplegia and hemianesthesia occurring upon the opposite side. When the lesion is high up in the pons, the facial paralysis, if it be present, will be upon the same side as the hemiplegia. If the sixth nerve be involved,

paralysis of the external rectus occurs, with internal strabismus of the eye upon the same side as the lesion. Vomiting, polyuria, albuminuria, and glycosuria are common.

Symptoms of Hemorrhage into the Medulla.—This is rare. If the hemorrhage be profuse, death rapidly takes place. The symptoms are similar to those of hemorrhage into the pons.

Symptoms of Hemorrhage into the Cerebellum.—The diagnosis of hemorrhage in this locality is very difficult. The onset is marked by pain in the occipital region and the back of the neck. Vomiting is a constant symptom. Convulsions also occur. There is motor paresis of the limbs upon the same side as the lesion, and, as a rule, the condition is fatal.

Symptoms of Ventricular Hemorrhage.—Usually coma is profound, and the condition generally terminates fatally. Ventricular hemorrhage may be suspected if symptoms of cerebral hemorrhage have occurred, in which partial recovery has taken place, followed by a sudden relapse into deep and profound coma. The temperature, which may have ascended slightly, falls rapidly, subsequent hyperpyrexia again being noted. The pupils may be either contracted or dilated.

Symptoms of Meningeal Hemorrhage.—There is marked pain in the head, rapidly followed by coma, with convulsions which are often localized early in the attack. Pain and convulsions are more common in this form of hemorrhage than in that of any other part of the brain.

Duration.—The initial coma may pass off in a few hours, the cases in which recovery is likely to occur being of this form. A favorable symptom is a return of the reflexes. There are, commonly, headache and some slight change in speech, which, however, may pass away, nothing but the paralysis remaining. Sudden death is quite rare. If recovery ensues, the leg is the first to regain power, the arm recovering later. The upper portion of the arm and the shoulder recover before the hand and forearm.

Secondary Phenomena.—If the paralysis persist for a long time, slight secondary changes develop; the leg, however, frequently regaining some muscular power. Later contractures and rigidity develop in the affected membranes, these being most pronounced in the arms. There may also be pain. Loss of power necessarily follows these contractures, and these changes are associated with sclerosis of the motor paths. The reflexes are exaggerated in this stage. Atrophy of the

muscles, tremor, arthropathies, and chorea may subsequently develop.

Prognosis.—In favorable cases the coma is rarely profound, some return to consciousness being noticed in from two to six hours. Cases in which no signs of consciousness are apparent after twenty-four hours are exceedingly unfavorable. Bradycardia, tachycardia, irregular pulse, slow or rapid respiratory movements, and especially Cheyne-Stokes respiration are unfavorable signs, as is also the appearance of sugar and albumin in the urine.

Treatment.—The patient must be placed in bed with the head elevated. If the arterial tension be high, venesection should be performed immediately. Ice-bags should be applied to the head. Croton oil or calomel are of great value in inducing free purgation. If the patient be unconscious for a considerable length of time, the bladder must be evacuated with the catheter.

Treatment of the Subsequent Condition.—The patient is necessarily confined to bed for some length of time, therefore attention must be given to the skin in order to prevent bedsores. The diet should be light and nutritious. Changing the patient's position from time to time is necessary to prevent hypostatic congestion. The bowels must be carefully regulated. Iodid of potassium and tonics are indicated. In some instances mercurials are beneficial. If a positive diagnosis of meningeal hemorrhage can be made, operative procedure must be considered. Massage and electricity are of benefit to the paralyzed muscles; these measures of treatment, however, should not be employed early.

THROMBOSIS AND EMBOLISM.

Thrombosis.—Thrombosis may develop from lesions of the blood-vessel walls, such as atheroma, the small branches being frequently the seat of the lesion; indeed, any roughening of the vessel wall may give rise to thrombosis, such as atheromatous plates, tubercles, various tumors involving the wall, and acute inflammations of the arteries. Thrombosis may occur secondarily from an embolus, or in some cases ligation of the carotid artery is followed by thrombosis that extends upward. In some instances an aneurysm is found to contain a large thrombus.

Changes in the constituents of the blood may give rise to

thrombosis, the following being the most important: In some of the primary anemias, particularly chlorosis; increased fibrin in the blood, this being specially noted in pregnancy; in some of the constitutional diseases, as gout, diabetes mellitus, syphilis; and in some acute infectious diseases, as diphtheria, enteric fever, and malignant endocarditis. Thrombosis may arise from blood parasites. Slowing of the circulation is a cause of thrombosis; it commonly accompanies edema.

Embolism.—Embolism is most frequently met with in the middle cerebral artery upon the left side. This may be due to the fact that an embolus finds its way through the left carotid artery more easily than through the right, this being the more direct course. Emboli most frequently arise from the breaking-up of a vegetation in valvular disease, mitral stenosis being the lesion which most commonly gives rise to it, or from a clot from an aneurysm, or atheromatous plates in a blood-vessel. The breaking-up of a thrombus may give rise to embolism. Fragments of bones, tumors, and parasites in the blood-stream may act as emboli. The affection is more frequent in females than in males, the greater number of cases arising from endocarditis.

Pathology of Thrombosis and Embolism.—The blocking up of a blood-vessel in the brain gives rise to an infarct, which may be swollen and anemic; or the area may be filled with blood, called a hemorrhagic infarct. If the embolus be infected, an abscess is liable to arise (metastatic abscess). The area in which the blood is cut off soon undergoes softening, and if much blood be contained in the affected region, it is called red softening. Later on, a fatty degeneration ensues and the part becomes yellow; this is known as yellow softening, and the latter condition may gradually merge into white softening, and the anemic infarct may degenerate and soften, constituting the principle of white softening. Inflammatory changes often develop around the area of infarction. The focus of infarction may gradually be replaced by fibrous connective tissue, or, in some instances, the degenerated material becomes liquefied and a cyst is noted in the area.

Symptoms of Thrombosis.—Prodromes are common; these consist in drowsiness and headache, the beginning of the attack being insidious. Hemiplegia occurs, which is also gradual in its onset. Ocular and other cranial nerve palsies may occur.

Symptoms of Embolism.—The symptoms of this closely

simulate cerebral apoplexy; indeed, it may be impossible to make a differential diagnosis. There is rapid loss of consciousness, with hemiplegia and disturbances of the pulse and respiration. A point of importance would consist in the fact that embolism is more frequent upon the left side of the brain than hemorrhage; hence, right-sided hemiplegia with organic valvular disease is in favor of embolism. Thrombosis has been noted most frequently in the middle cerebral artery, in the vertebral artery, in the basilar artery, in the internal carotid, and in the anterior cerebral arteries.

Prognosis.—Recovery from cerebral occlusion is slightly more favorable than cerebral hemorrhage. Complete recovery from the paralysis is exceedingly rare.

Treatment.—The treatment is the same as in cerebral hemorrhage

CEREBRAL ANEURYSMS.

Cerebral aneurysms of fair size occasionally involve the larger blood-vessels of the brain, particularly at the base. The middle cerebral on the left side is most frequently involved. Next in frequency are aneurysms of the basilar artery, the internal carotid coming next in order of frequency.

Etiology.—As in other blood-vessels, arteriosclerosis is perhaps the most important causative factor in cerebral aneurysms. Other causes may be embolism, trauma, and syphilis. Aneurysm is more frequent in the male sex, and after middle life.

Pathology.—The aneurysms vary greatly in size, from a miliary character to the size of a hen's egg, or even larger. They are commonly sacculated, but the fusiform and cylindric varieties have been noted. Rupture of the sac may take place, the blood extravasating into the brain-substance, and in some instances meningeal hemorrhage is encountered.

Symptoms.—The symptoms of aneurysm are chiefly those of a cerebral tumor. Headache is a constant symptom, and is often made worse by straining at stool or other muscular efforts. Vertigo, dizziness, and tinnitus aurium are common symptoms. Convulsions and vomiting are occasionally encountered. Rarely apoplectiform attacks take place, which may be due to a sudden distention or to a minute rupture. Optic neuritis is rare. Occasionally a sensation of pulsation that is apparent to the patient is noted, accompanied by sounds of which the patient is also aware. Careful auscultation of

the skull sometimes elicits a distinct bruit. Localization may determine the position of the aneurysm.

Diagnosis is difficult and often impossible.

Treatment.—The treatment is that of aneurysm in general. Internally, iodid of potassium should be given, especially as syphilis is so frequently a cause of blood-vessel disease.

ABSCESS OF THE BRAIN.

Etiology.—This condition is rarely primary; it is most often secondary to disease of the bones of the skull or soft tissues, such as ear disease, nose disease, and so on, to trauma of the skull, or to a septic infection carried from some other organ, most often the lung. The condition may be acute, subacute, or chronic. Abscesses rarely result from some of the infectious fevers. It occurs more frequently in males than in females, and most commonly between the ages of twenty and forty. It is more common in the working classes than in the well-to-do, probably because otorrhea is more likely to occur, head injury being more frequent among the poorer classes.

Pathology.—The abscess is most often situated in the temporosphenoidal lobe, the right side being more often involved than the left. The cerebellum is less frequently involved than the cerebrum, and still more rarely the pons and medulla. The reason for this common situation is on account of the relation to the internal ear. The abscess formation may be solitary or multiple. In the greater number of cases it is solitary. The pus may contain various pyogenic micro-organisms, and in rare instances the pus has been found sterile. The surrounding brain-structures may reveal the changes which are noted in acute inflammation, but in some instances where the abscess is of long standing, a thick layer of fibrous connective tissue is found. In the acute abscesses the pus is often blood-tinged, while in chronic abscess it is often thin and watery. The size varies greatly; it may be of any size from that of a pinhead to an abscess which in extreme cases involves nearly the entire brain.

Symptoms.—The symptoms are variable. Occasionally they are very severe, appearing rapidly, while in other cases the onset is insidious. The most characteristic symptoms are intense headache, vertigo, vomiting, mental dullness, and optic neuritis. There is a slow pulse early in the disease.

The pulse-rate in the greater number of cases is under the normal, indeed it is commonly between 40 and 60 a minute, and some cases have been noted in which the temperature is high and the pulse-rate 40 or 50 a minute. Rare cases have been recorded in which the pulse-rate ranged between 10 and 16 a minute. After surgical interference, in which the pus has been evacuated, the pulse-rate increases. Arhythmia is very common with this bradycardia. There are drowsiness and apathy. There are often gastric disturbances, such as anorexia, foul breath, heavily coated tongue, and constipation. In some instances muscular spasms, epileptiform convulsions, and paralysis such as hemiplegia and monoplegia occur. Motor and sensory aphasia and hemianopsia are present. Delirium is rare, but coma usually precedes the fatal issue. Occasionally there is marked and rapid wasting. There is much difference of opinion as to the range of the temperature. It not uncommonly clings to the normal point, and it also happens in many cases that it is subnormal. Occasionally some rise of temperature is noted—in many cases at the period of onset. If the condition be acute in its onset, fever is very often present. Some authors have stated that the temperature nearly always runs a subnormal course. It is probable that in such cases the course of the disease has not been closely observed, or that the condition has been attributed to other causes. Often at the close of the disease the temperature rises. Rupture of the abscess into the ventricles or inflammation of the meninges gives rise to fever. In the acute and subacute varieties fever may occur, but in the greater number of cases of this form of brain abscess the temperature is normal or subnormal. Respirations are usually slow, from ten to fifteen a minute. Cheyne-Stokes respiration occurs in some instances.

Prognosis.—The prognosis is grave.

Treatment.—The treatment is surgical. Early evacuation of the pus is indicated.

ENCEPHALITIS.

Inflammation of the brain, either local or diffuse, arises from some of the acute infectious diseases; from intoxications, such as food-poisoning, alcohol, nicotin, lead- or gas-poisoning, and also from injury. The most important infectious diseases giving rise to the condition are influenza, cerebrospinal fever, and

ulcerative endocarditis. It has been known to follow scarlet fever, measles, diphtheria, croupous pneumonia, hydrophobia, syphilis, pertussis, gonorrheal infection of the brain, tetanus, erysipelas, purulent otitis media, and acute anteropoliomyelitis.

Pathology.—Any portion of the brain may be involved. A point of frequent occurrence is in the gray matter: that is, in relation to the third and fourth ventricles and the aqueduct of Sylvius.

Symptoms.—As a rule, the disease begins acutely. There is either somnolence or coma from the onset, but this may be preceded by a stage of irritable jactitations resembling the onset of delirium tremens. Headache, vertigo, vomiting, and painful contractions of the muscles of the back of the neck are common. The pulse-rate is increased, as is also the respiratory frequency. As a rule, the temperature is normal or even subnormal. Rarely, fever is encountered. Incontinence of urine occurs in the terminal stages of the disease. Loss of appetite and constipation are common.

Duration.—The duration of the disease is from ten to fourteen days.

Prognosis.—The prognosis is unfavorable, few cases terminating in recovery.

Treatment.—Rest in bed is most important. The patient is confined to a dark room, and absolute quiet is essential. The head should be elevated; it should be shaved and ice-bags applied. Local blood-letting is important. Laxatives are extremely necessary: calomel should be given in large doses to the point of ptyalism. Iodid of potassium is valuable. If pain be severe, opium is indicated.

HYDROCEPHALUS.

Definition.—A collection of fluid within the ventricles or the subarachnoid spaces of the brain.

Etiology.—This condition may be either congenital or acquired. The acquired variety, sometimes called idiopathic hydrocephalus, is due to mechanical disturbances. The cause of congenital hydrocephalus is not known. It sometimes produces difficult labor. In congenital hydrocephalus the head is greatly enlarged and the bones of the skull are thin. The fontanels and the sutures may be large and the bones widely separated. Fluctuation may be detected. The face frequently appears small in comparison to the great enlargement of the

cranium. Upon examination of the brain in this condition it will be found that the ventricles are distended with fluid, which is usually clear. The brain-substance is flattened, and there is also fluid in the subarachnoid spaces.

Symptoms.—The symptoms of *congenital hydrocephalus* are difficulty in movements of the child on account of the weight of the head. There is impairment of intellect, but in some instances the mental capacity is good.

Acquired hydrocephalus may arise from a tumor at the base of the brain, giving rise to engorgement of the ventricular plexus of veins.

The symptoms vary. There may be headache and dimness of vision and the gait may become irregular. The pulse is usually slow. Many cases are difficult to diagnosticate.

Treatment.—Treatment is usually of no avail in this condition. Operative methods are advised by some authorities.

TUMORS OF THE BRAIN.

The following tumors have been found in the brain:

Glioma.—This tumor is limited to the central nervous system and the retina. It is usually solitary and quite firm, but in some instances it is soft and vesicular. These tumors may undergo softening and fatty degeneration, and a cyst may replace a great part of the new growth. The fluid is clear or turbid, and in some instances is black. These tumors vary greatly in size—from the size of a pea to that of a man's fist. The tumor may also undergo myxomatous degeneration, and is then known as gliomyxoma.

Neuroma may be found sometimes combined with glioma, when it is known as neuroglioma.

Sarcoma is quite common in the membranes, also arising from the bones of the skull, and in the adult this is a common form of brain-tumor. It may also be found in the pons. Many varieties of sarcomata have been encountered. Fibromata and osteomata are very rarely found in the brain. Adenomata and lipomata have been noted. Carcinomata may be either primary or secondary; in the greater number of cases they are secondary. Psammomata and cholesteatomata have been found. Gummata are commonly encountered in the brain.

Cysts of the Brain.—Cysts may develop from brain soften-

ing. Hydatids and cysticercus cellulosæ have been known to occur in the brain.

Symptoms.—As a rule, the symptoms of an intracranial growth are quite characteristic, but it must be stated that they may exist without giving rise to symptoms *intra vitam*. Generally, the characteristic symptoms consist in headache, optic atrophy, sensory disturbances, convulsions, vomiting, vertigo, and bradycardia.

Headache.—This is an almost constant symptom of cerebral tumor. It varies in severity, but, as a rule, is pronounced, increasing in intensity as the disease advances. It may be so severe that loss of consciousness due to the pain ensues. The pain is often described as diffuse, occupying the entire skull, or it may be deep-seated. It is often unilateral and localized to the temporal region. Pain which is constant in a local area in the head is always suggestive of an intracranial growth. The pain is increased by muscular movement, the use of alcohol, sneezing, excitement, change of posture, and so on. If pain be marked in the anterior portion of the head, it may extend down the face; and if it be felt in the back of the head, it sometimes radiates down the neck.

Optic Neuritis.—Double optic neuritis is very frequent in tumor of the brain; indeed, it is probably a symptom of more diagnostic value than headache. It may not be associated with a decrease in the field of vision, the latter often being normal. Single optic neuritis is less frequently encountered than double. When the neuritis is more pronounced on one side, it points to the fact that the growth is probably situated on the side showing the most marked inflammation of the optic nerve.

Disturbances of the Sensorium.—The patient is easily tired, any exertions readily fatiguing him. Numbness is complained of, especially in the skull, and it is difficult for the patient to express his ideas. He has a sleepy, tired expression; he answers slowly, questions having to be repeated several times before an answer is elicited. Finally, stupor and coma occur. The sphincters are relaxed. Melancholia is noted, and in rare instances talkativeness is encountered. Localized convulsive seizures occur, especially in cases in which the growth irritates and involves the cortex. The spasms may be confined to single muscle-groups, but may gradually extend and become general. Occasionally apoplectiform attacks are encountered. The status epilepticus is not infrequent. Vom-

iting takes place, especially when the base of the brain or the pons and the medulla are involved. As a rule, it is an early symptom, and is cerebral in character. It often occurs early in the morning, upon awakening, and may also occur with change of posture, without much nausea and retching, often with a clean tongue, and without influencing the appetite. It may be the precursor of an epileptiform attack.

Vertigo.—Vertigo may be present, most often as a temporary condition, and is due to disturbance of the cerebral circulation. If the vertigo be constant and severe, it is likely that the tumor involves the cerebellum.

Bradycardia.—In a limited number of cases the pulse may be slowed only for a brief time; bradycardia is, however, not so constant as a symptom of intracranial growth as it is in cerebral abscess. In some rare cases tachycardia has been noted.

Temperature.—As a rule, the temperature is normal or subnormal, fever being due to complications, particularly cerebritis and meningitis. If the tumor be situated in the basal ganglia, pons, or medulla, there may be hyperpyrexia. In some cases dyspnea is noted, as is also Cheyne-Stokes respiration. Yawning and hiccup (singultus) occur. Incoordination is rare; it is present when the growth involves the cerebellum.

Localizing Symptoms.—*The Motor Centers.*—If the tumor produce irritation of the lower portion of the motor area, spasm of the face or tongue is noted. Involvement of the middle portion will produce spasm of the arm, hand, or shoulder; and if the lesion be in the upper portion, similar symptoms develop in the leg, thigh, toes, and ankles. In some instances there are numbness and tingling, which are appreciated by the patient before the spasm begins. If portions of the motor area be destroyed, paralysis results in the muscle-groups that are in relation to those particular motor centers. Motor aphasia is produced by tumors involving Broca's convolution. The point of origin or signal symptom should be determined if possible, also the condition of the affected portion,—whether there be anesthesia or paralysis,—and also the character of the spasm.

Prefrontal Region.—In this region motor or sensory disturbances are often noted. Exophthalmos upon the affected side is observed in some cases.

Parieto-occipital Lobe.—Mind-blindness and word-blindness

are produced. The angular gyrus and the white matter beneath in some instances do not produce any symptoms.

Occipital Lobe.—Blindness, hemianopsia, and sometimes word-blindness and mind-blindness are noted when this lobe is involved.

Temporal Lobe.—If the lesion involves two-thirds of the posterior part of the first temporal convolution, and perhaps the second be involved, there is word-deafness, but sometimes a large tumor may exist in this area without giving rise to symptoms.

The Basal Ganglia.—Tumors situated in this area may produce hemianopsia, hemianesthesia, and in some instances hemiplegia. Optic neuritis and disturbances of the cutaneous and muscular ganglia are also noted. When the tumor involves the crura cerebri, ocular symptoms are pronounced, such as nystagmus and absence of pupil reflexes. There is ocular motor paralysis on one side when the third nerve is involved, and often hemiplegia of the opposite side.

Pons and Medulla.—The Pons.—Alternate paralysis may occur; that is, hemiplegia on one side, and the nerves involved on the opposite side. If the lesion be in the lower and inner portion of the crus, there will be paralysis of the third nerve and of the limbs, face, and tongue of the opposite side, known as the *syndrome of Weber.* If the sixth nerve be involved, there will be internal strabismus. If the seventh nerve be affected, there will be facial paralysis. Deafness follows lesions of the auditory nerve.

Medulla.—If the medulla be involved, paralysis of the nerves and hemiplegia may result. Tumors in this region also frequently produce vomiting, respiratory and cardiac disturbances, sometimes retraction of the head and neck, and difficulty in swallowing. If the cerebrum be involved, there may be symptoms of disturbed incoordination.

Prognosis.—The prognosis is always serious, with the exception of growths due to syphilis, when treated early. Surgical interference is sometimes beneficial in other varieties of tumors.

Treatment.—The patient must be placed in bed, and if possible absolute quiet maintained. The diet should be light and nutritious. The bowels must be carefully regulated, and full doses of iodids be given. If iodids do not relieve, mercurials should be tried; and if these measures do not afford relief, a surgeon should be consulted. If the tumor be local-

ized, surgical interference is often possible. Opium in some form is indicated to relieve the pain.

CEREBRAL PALSIES OF CHILDREN.

Two varieties of this affection have been observed—that which is noted at birth being called *birth palsy*, and one which occurs some time after birth, being preceded by health, and called *infantile hemiplegia*. The former usually involves both legs or both arms, while the latter is usually hemiplegic.

BIRTH PALSY.

Etiology.—This disease may arise from abnormalities of labor or may have its origin during intra-uterine life. It is due either to disease of the mother or to abnormalities of the fetus when occurring in intra-uterine life.

Difficult labor is an important cause, or, in some cases, a precipitate labor. Commonly, it arises in those instances in which forceps are employed. Young primiparæ are not so liable to give birth to children suffering from this affection as older primiparæ. Birth palsies are more frequent in males than in females. Syphilis seems to play a part in the production of this disease.

Pathology.—A lesion commonly present is hemorrhage in the membranes, giving rise to pressure upon various brain-centers. If there be pressure upon the third frontal convolution, disturbances of speech are noted, and if the lesion extend forward, there may be disturbances of the muscles of the eye; hence, squint may be encountered. If the disease has persisted for some time, the brain is frequently found softened (porencephalus) or there may be sclerotic changes.

Symptoms.—The symptoms are those of weakness associated with stiffness, which may involve both legs or both arms, sometimes both legs and one arm, and occasionally one arm and one leg on the same side. The gait is spastic if the legs be involved, the patient dragging the affected member. In some instances ankle clonus is present. The knee-jerks are exaggerated. Walking may be entirely interfered with, and spasm may develop. A tremor may be present. The ocular muscles are sometimes paralyzed, either singly or in groups. In some children the mental condition is very much dis-

turbed, while in others it is good; indeed, in some instances it may be acute. Speech may be disturbed. Convulsions which have a tendency to recur are noted. Such deformities as talipes and curvature of the spine often develop as the child grows older.

Prognosis.—The prognosis is unfavorable, although it must be said that some cases improve. The usefulness of the arms and legs is often much impaired, and if there be mental disturbance, there is little hope for improvement.

Treatment.—Careful hygienic measures are important. As the child grows older systematic exercise and massage are useful. Tonic treatment is quite important. The food should be nutritious and easily digestible. If possible, deformities should be prevented.

INFANTILE HEMIPLEGIA.

Etiology.—This occurs before the sixth year, most frequently during the first two years of life.

Pathology.—There is still much discussion as to the lesion. Two views are held—one by Strümpell, who believes that the affection is the result of an acute inflammatory condition of the gray matter of the cortex; and, second, there is a view held by Gowers, who believes that it is due to vascular obstruction.

Symptoms.—The onset is frequently marked by general malaise and slight fever, which terminates in a convulsion, the fit beginning upon the side which is afterward paralyzed. Convulsions recur at short intervals. The tongue is often bitten, the sphincters are relaxed, and consciousness is lost during the fit. Rarely there is no convulsion and only momentary loss of consciousness. The paralysis, as a rule, involves the face, arm, and leg, and is most marked in the arm, while the face suffers but slightly. As the child grows, the parts fail to develop; this hypoplasia being a striking feature of the disease. The knee-jerk is increased, and ankle clonus may be present. In some instances there is a peculiar tremor (chorea spastica), which is produced during voluntary movements. Mental weakness may follow the condition.

Prognosis.—The prognosis is good as regards life. Mental impairment and physical debility persist, but occasionally improvement is noticed in the affected paralyzed members. Impairment of speech rarely persists.

Treatment.—Hygienic measures are of the greatest importance in the treatment. Systematic exercise and massage are indicated. During the convulsions inhalations of small quantities of chloroform are frequently effective. After the attack a course of bromids is useful.

GENERAL AND FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

EPILEPSY.

Definition.—Epilepsy is a disease of the nervous system, characterized by loss of consciousness, with or without convulsions. It is a common disease, and it is estimated that 1 in 500 persons suffers from it.

Synonym.—Falling sickness.

Etiology.—Heredity plays an important part in the production of this disease. Gowers, in an analysis of 1450 cases of epilepsy, found a family history of the disease in two-thirds, and a previous history of insanity in one-third of the cases. In neurotic families the females are more likely to suffer than the males. Alcoholism and syphilis are important predisposing causes, in as far as heredity is concerned. The majority of cases begin before the twentieth year of life. Injury to the head may produce epilepsy, as do organic lesions of the brain-substance, such as thrombosis or embolism. It may follow the infectious diseases, especially scarlet fever, cerebrospinal fever, measles, and enteric fever. It sometimes follows the prolonged use of some drugs, as alcohol, lead, antipyrin, and cocain. The retention of putrefactive products in the gastro-intestinal tract, giving rise to auto-intoxication, may cause epilepsy. Sunstroke, sexual excesses, menstrual irregularities, and masturbation have been noted as causative factors. Occasionally, epilepsy is said to be due to reflex causes, such as intestinal worms, old cicatrices, nasopharyngeal disease, and eye and ear affections. Mental emotion and anxiety must also be mentioned as exciting attacks in those who are affected with the disease.

Symptoms.—The symptoms vary; they first begin with premonitory signs, so that the patient may be able to tell that

an attack is coming on. These premonitory symptoms are known as the *aurœ*. The auræ may consist of a feeling of uneasiness, with restlessness, irritability, and anxiousness. There may be fear or emotion, the patient having queer ideas, and a feeling that something is going wrong. Sometimes insanity may precede the attack (preepileptic insanity). On the other hand, flashes of light may occur and a certain color may appear before the eyes, or there may be actual loss of sight. Visions may appear, which may be either pleasant or disagreeable. Loss of hearing is not uncommon, or there may be peculiar sounds, such as hissing, whistling, or musical. Occasionally disagreeable odors occur, or there may be a peculiar taste in the mouth—salty, bitter, sour, or sweet. There may be a sensation of numbness and tingling in the extremities, which usually rises toward the head; sometimes this sensation begins in the thumb, fingers, wrist, or foot, and travels upward. In other cases there is discomfort in the epigastric region, this even amounting to severe pain, which progresses toward the head. When it reaches the throat, there is a sensation as if there were a ball situated there. This may be associated with nausea, vertigo, and headache. Occasionally muscular movements, twitchings, etc., take place. There may be paralysis, and, finally, there are sensations of chilliness or heat, and congestion or sweating of the extremities. In many instances the aura is absent altogether.

The Attack.—The attacks are divided into two principal classes—the severe or major epilepsy (*grand mal*), and the light or minor attacks (*petit mal*).

Major Epilepsy.—The attack often begins with a loud cry (the epileptic cry), the patient falling into unconsciousness, and into a state of tonic spasm. The face often becomes cyanotic, the head may be thrown backward or rotated to one side, the spine is often curved backward, and the legs are extended. The phalanges are often flexed and the thumbs drawn into the palms. The jaw is locked and the spasm may be so powerful as to dislocate certain joints. Soon twitchings take place in the muscles of the face, neck, and extremities. The tongue may protrude between the teeth and is often bitten. The pulse-rate is quickened, the respiration is rapid, the pupils are dilated and do not react to light. If the convulsions are long continued, the temperature may rise to 105° F. or over; otherwise the temperature remains normal. The body is covered with

cold sweat, and involuntary evacuation of urine and feces is common. The symptoms gradually ameliorate, the patient recovers consciousness or falls into a deep sleep, which may last from several minutes to several hours.

Minor Epilepsy.—There may also be many variations in this form of epilepsy. It is characterized by momentary loss of consciousness without convulsions. Occasionally there may be slight twitching of the muscles. Rarely the patient falls to the floor, but recovers immediately. The attack is brief, the patient recovering almost momentarily. Pallor of the face and involuntary evacuation of urine sometimes occur. Both forms, major and minor epilepsy, are sometimes replaced by conditions of unusual excitement or depression, which is called *psychic epilepsy.* This may last for an hour or more, in which the patient may develop homicidal tendencies. In some cases a prolonged sleep may take the place of the psychic equivalent.

Postepileptic Symptoms.—As a rule, there is more or less headache, but the attack may terminate without further symptoms. After a severe paroxysm the reflexes are usually diminished or absent. Later this follows a period of excitement, in which the knee-jerks are exaggerated and ankle clonus occurs. In the majority of cases the patient goes into a prolonged sleep. Rarely great hunger develops after an attack. Occasionally insanity may result. The patient may have but one seizure a year, or one a month, or he may have several in a day. Occasionally attacks of epilepsy follow each other so rapidly that there appears to be no interval of rest. This is known as the *status epilepticus.* When this occurs, death is not uncommon. Occasionally the entire attack takes place at night, while the patient is sleeping. This has been termed *nocturnal epilepsy.*

Course.—The disease is extremely chronic, and the tendency is to an increase rather than to a decrease of the attacks. Insanity develops in 10% of the cases, and some mental impairment occurs in many well-marked cases. It may be stated in general terms that the average length of life is shortened by epilepsy.

Diagnosis.—In a well-marked case the diagnosis is easy; it is only in ill-defined cases that difficulty arises.

A differential diagnosis must be made between epilepsy, syncope, and hysteric attacks.

Epileptic Seizures.[1]	*Fainting Attacks.*
Absence of exciting causes.	Exciting causes in the way of hot rooms, bad air, emotional strains.
Aura of brief duration or absent.	Premonitions for some time before loss of consciousness.
Sudden loss of consciousness.	Gradual loss of consciousness.
Pulse normal.	Pulse weak, often scarcely perceptible.
Pupils dilated and light reflex lost.	Pupils small or unchanged.
Spasm tonic and clonic.	No spasm.
Often involuntary micturition and defecation.	Rarely or never.
Biting of the tongue.	No biting of the tongue.
Cyanosis.	Pallor.
Short duration of unconsciousness.	Duration of unconsciousness longer.
Automatism or stupor after attack.	Speedy recovery after attack.

Epileptic Seizures.[1]	*Hysteroid Attacks.*
No exciting cause.	Emotional cause.
Aura or no premonition.	Globus, palpitation, malaise.
Epileptic cry.	Crying, talking, screaming during attack.
Sudden and complete loss of consciousness.	Loss of consciousness incomplete.
Pupils dilated.	Pupils unaltered.
Tonic followed by clonic spasm.	Rigidity, opisthotonos, struggling and tossing movements.
Biting of the tongue.	Biting at self or others and objects at hand.
Involuntary micturition and defecation.	Never.
Duration brief.	Duration often for long periods.

Prognosis. — The prognosis as to cure is unfavorable. Under the most favorable circumstances it has been estimated that only from 2% to 6% recover. The treatment may relieve the patient and hold off the paroxysms for some time. The more favorable cases for cure are in those of epilepsy arising in adult life.

Treatment.—The physician should endeavor to determine the cause of the illness, which should be removed if possible. It is best for the epileptic to be treated in an epileptic colony in which he may have out-door occupation. The general health must be carefully looked after. The bowels should be kept open and the diet regulated. The diet should consist of meat, fruit, cereals, and vegetables, meat being sparingly partaken of and only once a day. Cold sponge-baths (60° F. to 70° F.), twice daily, are of decided benefit. The medicinal treatment is chiefly empirical, the best results having been obtained by the use of the bromids. A combination of some one of the bromids with antipyrin is favored by some practitioners. Surgical interference as a cure for epilepsy, such as excision of

[1] From Loomis-Thompson.

a cortical focus, or opening of the membranes, has not proved successful.

JACKSONIAN EPILEPSY.

Definition.—A disease of the nervous system characterized by convulsions, and due to some irritative lesion of the cortical motor centers of the brain.

Synonym.—Cortical epilepsy.

The affection is due to an irritative lesion of the cortex of the brain which may destroy the region in which it is situated. The irritation may be due to an exostosis, a depressed fracture, tubercular and other tumors, meningitis, and especially syphilitic disease of the cortex.

The lesion tends to destroy the affected area, giving rise to paralysis with secondary degeneration. If this affect particularly the face, arm, or leg, the position of the lesion can be determined with more or less accuracy.

The symptoms consist of spasm, which, as a rule, is local in character. It is always so at the commencement of the affection, but as the disease advances and spreads the spasm (convulsion) may become general. As a rule, and especially in the milder forms, consciousness is preserved. Sometimes tingling and other sensory phenomena may precede the attack.

The treatment is surgical.

INFANTILE CONVULSIONS.

Synonym.—Eclampsia.

Convulsive attacks similar to those of epilepsy are not infrequent in children. The attacks differ, however, in the fact that when the cause is removed, there is no return.

Etiology.—Gastro-intestinal disturbances, such as enterocolitis, which give rise to debility cause convulsions in children. Irritations due to dentition, overloading the stomach, intestinal parasites, otitis, and phimosis produce the disease. In the course of rickets convulsions are not uncommon. The acute infectious diseases often begin in children with convulsions, particularly croupous pneumonia, scarlet fever, and measles. Congestion of the brain occurs during the course of whooping-cough, and may produce the condition; and diseases of the nervous system, such as infantile hemiplegia, meningitis, tumors, and other brain lesions, cause the

affection. Occasionally convulsions occur at birth, and may persist for weeks or months.

Symptoms.—The attack comes on suddenly, occasionally being preceded by restlessness, grinding of the teeth, or muscular twitchings. The spasm may begin in the hand, most often in the right hand. The eyes stare and are fixed, the body becoming stiff, and the respiration may be suspended, causing congestion. Clonic spasms commonly follow. When the attack subsides, it is often followed by sleep or the child may become stupid and pass into coma. As a rule, when the attacks are due to indigestion, the convulsion is single. In rickets and intestinal disorders several convulsive seizures may follow one another. If the attack is limited to one side, there may be slight paresis ; but if the attack be due to infantile hemiplegia, complete paralysis follows. During the attack the temperature is raised.

Prognosis.—When the convulsion follows intestinal disease, the prognosis is unfavorable. Convulsions occurring in the acute infectious diseases and those resulting from intestinal and peripheral irritation are not so severe, recovery often taking place.

Treatment.—The cause of the convulsion should, if possible, be removed. If it be due to an overloaded bowel, an emetic or a purge should be promptly given. If it is the result of undigested food, an emetic may often be employed. If the child be teething and the gum looks abnormal, it should be lanced. During the convulsion the child should be placed in a warm bath (about 95° F.), and cold compresses or cold water applied to the head. During the attack chloroform may be given by inhalation. If the convulsions occur after the child comes out of the chloroform narcosis, opium should be given with great caution. Amyl nitrite may be used by inhalation or chloral may be given by the bowel. After the attack a course of bromids is useful. Every effort should be made to preserve the nutrition of the child.

CHOREA.

Definition.—Chorea is a disease of the nervous system, characterized by involuntary contractions of muscles or muscle groups, accompanied by weakness and often by slight mental derangements.

Synonyms.—Chorea minor; Sydenham's chorea; St. Vitus' dance.

Etiology.—Chorea is a disease of childhood, although it may occur at any age. Between the fifth and the fifteenth years is the age at which the affection most commonly develops. Females are more apt to be affected than males, and it is very likely to occur in children who are descendants of neurotic parents. The disease is more common in temperate climates, and it affects particularly the white race, the negro and the Indian having some degree of resistance. It is most prevalent in the spring months. Acute rheumatic fever has been noted as an exciting cause in over 20% of the cases. School life, fright, shock, worry, and strain have been noted as predisposing factors. It occasionally results from reflex causes, such as intestinal worms, eye-strain, nasal disease, and sexual disorders.

Pathology.—The pathology of chorea is still very obscure, and no constant lesions are encountered. By some it is believed to be a functional brain disturbance affecting the centers which control the motor apparatus, while others believe that it is due to embolism, as emboli have been found in some of the smaller vessels of the brain. Some observers hold the view that it is an infection, as it is so frequently associated with acute rheumatic fever and endocarditis.

Symptoms.—The disease begins with feeble involuntary twitchings of the muscles of the face or hands. In the beginning the affection is most often unilateral. Twitchings of the mouth, jerking of the head, and winking are all common. Some loss of power is found in the hand, as the patient drops articles when the involuntary spasm comes on. The patient can not sit still; the shoulders are twisted; walking is sometimes rendered difficult, and the patient is apt to stumble. Soon both sides of the body become implicated, and the involuntary movements increase, but they are usually marked upon the side first affected. There may be twitching of the tongue and lips, speech becoming affected, and deglutition may be interfered with. As a rule, the movements cease in sleep, but in severe cases the muscular contractions may continue during sleep and interfere with the patient's rest. The child becomes restless, irritable, and peevish. There is emotional disturbance and sometimes dullness. The appetite is interfered with, and some anemia and loss of flesh are noted. Occasionally nocturnal enuresis occurs. The specific gravity of the urine

is high, there being an excess of urea and phosphates. There are no reactions of degeneration. The pulse is frequent—from 100 to 130 a minute, even without a cardiac lesion. In some cases there is a blowing murmur heard over the heart, which may be either functional or organic. In from two to five weeks the acme of the disease is reached. As a rule, it continues for several months or longer. In rare instances the affection may be limited to one side; it is then known as *hemichorea*. In other cases the twitchings may be slight, but the motor symptoms very marked; this condition is rare, and has been termed *paralytic chorea*.

Prognosis.—The disease is rarely fatal except in those cases in which great mental excitement occurs with delirium, hallucinations, and illusions. This has been called *chorea insaniens* or *maniacal chorea*. Relapses are common, the average number being two.

Treatment.—If the child attends school, it had better be taken away and its studies discontinued. Rest in bed with long sleeping hours is of marked benefit. In the severer cases the child must be kept constantly in bed; in other cases moderate exercise in the open air is of use. Sponging with cold water, especially the back, chest, and neck, is of great benefit. The diet should be simple—meats and highly seasoned foods being excluded. Arsenic, antipyrin, bromid of zinc, and quinin are the most useful drugs.

CHOREIFORM AFFECTIONS.

THE SPASMODIC TICS.

Various forms of spasm have been described resembling chorea. These are chronic in nature, the spasm often being violent, the disease lasting for many months or years. The following forms have been described: (1) Habit-spasm or habit-chorea; (2) spasmodic tic, including wry-neck and mimic spasm; (3) psychic tic.

Etiology.—The disease almost always occurs in childhood, between the fifth and the fifteenth years. Males are more frequently affected than females, the child most often being of a neurotic temperament. The disease may follow an attack of ordinary chorea. Occasionally overwork, fright, injury, or shock may act as predisposing causes. Masturbation is also an etiologic factor.

Symptoms of Habit-spasm.—These consist of an irregu-

lar twitching of the facial muscles, particularly those of the mouth and eyes. A constant snuffling of the nose is also common. The muscles of the shoulders often jerk, as may also those of the arms and legs.

Spasmodic Tic Proper.—As soon as the spasm becomes localized, one or several nerve-centers becoming involved, it is known as spasmodic tic. Of this there are many clinical varieties. A common type is where the muscles of the larynx are affected. This is known as *laryngeal tic*, or chorea of the larynx. At intervals the patient may whistle, bark like a dog, or the cry may resemble the epileptic cry, and there may be cough and frequently repeated hacking. The spasms are increased under excitement, and they disappear during sleep. The muscles of the trunk may be affected so that the gait becomes awkward.

Wry-neck.—In some cases there is a tonic spasm of the sternocleidomastoid and trapezius muscles.

Facial Tic.—Tic douloureux is occasionally associated with spasm of the muscles of the face, known as *facial tic.* It is most common in advanced life, occurring particularly in women.

Jumping Disease (*Myospasmia; Gilles de la Tourette's Disease*).—This is a curious form of convulsive spasm in which involuntary explosive utterances, which are frequently profane, occur. It has shown itself in many countries. The disease begins in the child between the ages of five and sixteen years, and is more prevalent in the male than in the female sex. The disease occurs in neurotic families, often being hereditary. In this country those affected with the disease are often called the "jumpers."

Prognosis.—The prognosis is good, as a rule. The spasmodic tics occurring in the muscles of the neck, face, and trunk are not so favorable, and the psychic tics generally are incurable.

Treatment.—The treatment consists of the same therapeutic measures that are carried out in chorea proper.

HEREDITARY CHOREA, OR HUNTINGDON'S CHOREA.

This is a very rare disease. It is always hereditary and most frequently by direct transmission. The disease is transmitted through the sexes equally. Both sexes are equally affected. No exciting cause has been discovered.

The symptoms consist in slight jerking movements of the

facial muscles or of the muscles of the extremities. The patient makes curious gestures, the gait is interfered with, the muscles of the face being particularly affected. With all this there is marked interference with the mental processes. At first loss of memory is slight, but later it becomes so marked that the patient can not attend to his ordinary occupations. Periods of excitement occur, rarely, however, with active mania or with homicidal or suicidal tendencies; occasionally acute mental symptoms develop. Melancholia is a prominent symptom. So few cases have been reported that the opportunities for postmortem studies have been necessarily limited. The process appears to affect primarily certain groups of nerve-cells with degenerative changes and meningeal thickening.

Prognosis.—The disease is fatal, although the course may be prolonged.

Treatment.—The treatment is purely symptomatic and prophylactic. Choreic individuals should not be permitted to marry or intermarry. No drugs have been described that have any especial effect.

MIGRAINE.

Synonym.—Hemicrania.

Etiology.—Women suffer to a greater extent than men in the proportion of three to one. The disease often begins in childhood, and is rare after fifty; it shows strong hereditary tendencies. Digestive disturbances are frequently associated, and the disease occurs in neurotic females.

Symptoms. — The principal symptom is the headache, which shows more or less periodicity, the patient in the interval often enjoying perfect health. At the onset there are often prodromes, such as a feeling of uneasiness or languor. These symptoms are followed by pallor and some vasomotor spasm. As the headache becomes severe, flushing of the face succeeds the pallor. The disease is, as a rule, unilateral, the left side being oftener affected than the right; both sides of the head may be affected. The pupil upon the affected side is often smaller, and the eye may be retracted. Often permanent local grayness of the hair is noted upon the affected side. There is commonly disturbance of vision, the duration, however, being temporary. There are flashes of light, blurring of the sight, hemiopia, and pho-

tophobia. Occasionally there is tinnitus aurium associated with vertigo. In the majority of cases the symptoms are accompanied by marked gastric disturbances, such as nausea, with frequent vomiting. The retching often becomes extreme and aggravates the headache. The urine passed during the attack is high colored, being rich in solids.

Prognosis.—The prognosis is favorable, although the attacks recur and may become more frequent.

Treatment.—The treatment consists in the management of the attack and the prevention of the recurrence. Many drugs have been advised for the relief of the pain. Ergot in full doses has been recommended. Some of the coal-tar products, such as antipyrin, antifebrin, and phenacetin, are of use, especially if administered early. The use of full doses of salicylate of sodium is valuable in some cases, while in other instances the bromids, with caffein, are of benefit. Gelsemium has also been highly recommended. Underlying conditions, such as lithemia, the gouty diathesis, digestive disturbances, and constipation, should be looked after. As a prophylactic, nitroglycerin in $\frac{1}{100}$-grain doses, taken after meals, with bismuth and pepsin, has been said to be of value. Mild purging with calomel from time to time often prevents attacks.

PARALYSIS AGITANS.

Synonyms.—Parkinson's disease; shaking palsy.

Etiology.—The disease is one of advanced life, beginning, as a rule, between the ages of forty and sixty. Men are twice as often affected as women. The influence of heredity has not been definitely determined. Great stress has been laid upon fright, mental emotion, trauma, and illness as etiologic factors. It does not appear that alcohol, tobacco, and lead are causative factors.

Pathology.—The pathology of this disease is still obscure. Some believe the affection to be a functional disorder, while others believe that the lesion is in the medulla, pons, or spinal cord.

Symptoms.—The most important symptom is tremor. As a rule, this begins in one extremity, most often the hand, and then spreads to the leg of the same side of the body, later to the arm and leg on the opposite side. At first the tremor is slight, becoming more marked as the disease advances; the rate varies from three to five vibrations a second. Under

rare circumstances the tremor may continue during sleep. The tremor ceases temporarily during voluntary motion; however, it increases after a short period of rest. Tremor of the head, which was supposed by Charcot not to take place in this disease, has been noted by many observers. After some little time muscular rigidity and weakness occur in the affected muscles, giving rise to a characteristic attitude. The head is bent forward, the shoulders are stooping, the thighs are adducted, and the knees are more or less flexed. The elbows are slightly bent and the wrists extended, the face often becoming expressionless. The reflexes vary; they are increased in some cases and diminished in others. The gait is often affected in a peculiar manner. The steps are short, the patient appearing to run; this is known as *festination* or *propulsion*. The voice is often of high pitch, and the articulation may be interfered with. Mental symptoms occur in some of the cases, such as depression and loss of memory. Tachycardia has also been observed.

Prognosis.—The disease is incurable, lasting for years.

Treatment.—The treatment is symptomatic. Occasionally hyoscyamin or hyoscin controls the tremor. The general hygiene of the patient should be looked after. Tonics, such as arsenic and strychnin, are of use.

FACIAL HEMIATROPHY.

Synonyms.—Neurotic atrophy of the face; hemiatrophia facialis progressiva.

Etiology.—This is a rare disease, found most frequently before the age of twenty, being exceedingly rare after thirty years of age. It is much more commonly found in the female than in the male sex. Heredity seems to play some part as a predisposing cause. It has been known to follow the acute infectious diseases. Trauma has also been noted as a predisposing factor. In many cases no apparent cause is discernible.

Pathology.—A few cases have been observed upon the postmortem table. In Mendel's case interstitial neuritis of the trigeminus and its root was found.

Symptoms.—Usually the first thing noticed is the appearance of a whitish or yellowish patch upon the cheek, chin, or forehead. This gradually increases in extent. In some instances several patches appear at the same time, which finally

coalesce. The skin in this area becomes glossy or parchment-like, and a depression may form which is due to atrophy of the subcutaneous fat. The rapidity with which these changes occur varies in individual cases. It is most common upon the cheek. The atrophic process may involve the hairy parts of the face, in which case the hair becomes thin and finally falls out. Sweating is lessened upon the affected side, and may finally disappear entirely. The muscles of the face become atrophied; no paralysis, however, is noted. Atrophy has also been observed in the tongue. In long-standing cases the bones of the face—the frontal, the malar, and the superior and inferior maxillary—become atrophied. The nasal cartilages and even the ear may be involved. Sensation is rarely affected. Associated symptoms are neuralgic pains in the distribution of the fifth nerve and twitching of the muscles. In some cases the atrophy of the alveolar process becomes so marked that the teeth become loosened and drop out.

Prognosis.—The disease is progressive. It may, however, be spontaneously arrested in some instances but it does not shorten life.

Treatment.—General tonics, such as quinin, iron, arsenic, and strychnin, are useful. Gentle massage and the constant electric current are of value in allaying pain.

ACUTE CIRCUMSCRIBED EDEMA.

Definition.—A disease characterized by local edema, showing a tendency to recurrence at definite intervals.

Synonyms.—Angioneurotic edema; acute inflammatory edema.

Etiology.—It may follow severe physical or mental exertion or exposure to cold, and may be associated with malaria, the abuse of alcohol and tobacco, and certain diseases of the nervous system. It is met with in hysteria, neurasthenia, and exophthalmic goiter. Heredity seems to play some part in the causation of this disease.

Symptoms.—The onset of the disease is sudden, and is characterized by itching and swelling, the face being most frequently attacked, although sometimes the extremities and trunk may be affected. The mucous membranes are sometimes involved, so that if the gastro-intestinal mucous membrane be the seat of the disease, there may be vomiting, colic, and diarrhea. If it involve the pulmonary structure, hemoptysis may occur.

Hematemesis and hemiglobinuria have also been noted. The swellings rarely pit upon pressure unless they have lasted for some time. The edema lasts from a few minutes to several hours, then disappears, and recurs at certain definite intervals, which may be days or even months. The edematous area is of a rose-red or dull whitish color, the area being distinctly outlined. It is believed that the disease is due to a vasomotor neurosis.

Treatment.—If possible, the predisposing cause should be removed. If there be marked pain, opium is indicated. Local soothing applications are grateful to the patient. During the interval quinin and atropin are of use in preventing the recurrence.

RAYNAUD'S DISEASE.

Definition.—Raynaud's disease is characterized by an alteration in the blood-supply with a disturbance of nutrition of the extremities and internal structures, which is, as a rule, symmetric. The disease was first described by Raynaud in 1862.

Synonyms.—Local syncope; local asphyxia; symmetric gangrene.

Etiology.—The disease occurs at all ages; it is, however, more frequent in middle life, and is more often found in females than in males, and most individuals affected are descendants of neurotic families. Syphilis, acute rheumatic fever, rachitis, and various forms of anemia have been mentioned as predisposing causes. The acute infectious diseases, such as influenza, enteric fever, and also trauma, especially injury to the nervous system, are mentioned as exciting causes. In some cases there has been a history of great fatigue, fright, and mental emotion.

Three **varieties** or degrees of intensity of the disease are described: (1) Local syncope; (2) local asphyxia; (3) symmetric or local gangrene.

Local Syncope.—The peripheral parts of the fingers or toes and sometimes the entire hand or foot presents a white and glossy appearance, following fatigue or exposure. The area is cold, and there is a deficient blood supply. The lobes of the ears and the tip of the nose are sometimes involved. The duration is variable, lasting from minutes to days, rarely longer than a few hours. After the pallor subsides a reaction sets in, the skin becoming red and hot.

Local Asphyxia.—If the condition just described continues, the fingers, the toes, and the hands become congested and swollen. Pain, ensues, but it is not intense. The condition sometimes arises without the stage of local syncope, and in some instances there are very severe pain and anesthesia of the affected area. Constitutional symptoms are not marked. Attacks are produced by the slightest exposure, and may recur a number of times.

Symmetric or Local Gangrene.—The fingers or, in some instances, a single phalanx of a finger may be the seat of the affection. Sometimes the toes are affected in a similar manner. Local gangrene may follow local asphyxia. The gangrenous part presents the common characteristics that are noted in such an area, being deeply discolored. Blebs are seen, and a line of demarcation plainly shows the boundary between the necrotic and the healthy tissues. The fingers and toes may slough off and a cicatrix form. In some instances the tip of the nose is involved, and in more extensive cases the limbs are affected. In rare cases even the trunk may be involved, there being patches of gangrene scattered here and there. In some instances there is extensive multiple gangrene. The symptoms most commonly associated with Raynaud's disease are hemoglobinuria, unconsciousness, or mental torpor, and sometimes peripheral neuritis.

Prognosis.—The disease may recur in adults from time to time. Unless there be some underlying chronic ailment, as syringomyelia, tabes, or tuberculosis, the prognosis is favorable.

Treatment.—A change of climate is advisable in many cases. The local treatment consists in rest of the part. It should be wrapped in carded wool. Irritants or lotions should not be applied locally as they often increase the tendency to gangrene. Nitroglycerin and amyl nitrite are recommended by some. Opium must be administered when the pain is severe.

ERYTHROMELALGIA.

Definition.—"Erythromelalgia is a chronic disease in which a part or parts of the body—usually one or more extremities—suffer with pain, flushing, and local fever, made far worse if the parts hang down" (Weir Mitchell).

Synonym.—Red neuralgia.

Etiology.—The majority of cases occur during the middle

periods of life, some few cases having been recorded between the ages of sixteen and twenty-one. The disease is most common in the male sex. It is supposed that occupations which require standing and exposure to varying temperatures predispose, such as iron-workers, engineers, seamen, letter-carriers, and so on. It appears from cases that have been recorded that previous ill health has an important bearing as an etiologic factor, as do also syphilis, abuse of alcohol, and trauma.

Pathology.—The pathology of erythromelalgia is not definitely determined. Most observers believe it to be a peripheral irritation of the nerve-endings themselves, or primary inflammation of the tissues around the nerves, while others hold that the condition starts centrally.

Symptoms.—The affection commonly begins with a burning pain, usually in the sole of one foot, which is aggravated by standing and voluntary movements, being relieved in the recumbent posture and when the part is elevated. After rest the pain returns, and subsides less easily than upon each former occasion. The pain is soon followed by redness in the affected area, which is at first circumscribed, or several areas may form at the same time, which soon increase in extent. The veins become distended, and pulsation is noticed in the arteries, the pain constantly increasing in severity. Sensibility to heat and cold is increased in the affected part; the reflexes, however, remain normal. The surface temperature shows increased heat in the part. There is some swelling and the part may pit upon pressure. Cold and cold applications relieve the pain, whereas heat increases the severity of the paroxysm. Relapses are common. The attacks may last from several hours to many years. By some authorities it is believed that this disease is a variety of Raynaud's disease (local syncope).

Treatment.—The food should be nutritious and should be freely given. Rest is the most important factor in the treatment, the limb being placed in the horizontal position. Faradism is useful in the milder cases. Local applications of cold afford relief.

MÉNIÈRE'S DISEASE.

Definition.—A disease characterized by vertigo, tinnitus aurium, deafness, and vomiting, and due to disease of the internal ear.

Synonyms.—Aural vertigo; labyrinthine vertigo.

Etiology.—The disease is rare before the thirtieth year of life, being more common in the male than in the female sex. It is supposed that gout, syphilis, degenerative changes due to age, and hemorrhage into the middle ear are causative factors. An attack may be brought on by gastric disturbances, cerebral or other irritations.

Pathology.—It is probable that the symptoms of this disease are due to disturbed function of the peripheral or central portions of the vestibular nerve or of the organs in relation to it.

Symptoms.—The important symptom of the attack is vertigo, which varies considerably, being slight in some instances and so severe in others that the patient is compelled to seek the recumbent posture. In some cases the patient may fall abruptly to the ground. These attacks occur in paroxysms at intervals of days, weeks, or months. In the severer cases nausea and vomiting, and sometimes unconsciousness, occur coincidently with the vertigo. Optic phenomena, such as nystagmus and diplopia, sometimes appear. Tinnitus aurium is usually constant, becoming worse during the paroxysm. Deafness is present during the attack, but usually disappears during the interval.

Prognosis.—The attack may prove fatal, but in the majority of instances improvement and even complete recovery occur.

Treatment.—The bromids are useful. A counterirritant over the mastoid process of the temporal bone has proved efficient in many instances. It is always necessary to treat the underlying condition. Quinin and salicylate of sodium in large doses have been advised. Nitroglycerin and the nitrites are valuable if arteriosclerosis be present.

OCCUPATION NEUROSIS.

Definition.—This neurosis is due to the constant use of certain groups of muscles in occupations which necessitate delicate movements, producing cramp, spasm, paralysis, tremor, or neuralgic phenomena.

Synonyms.—Fatigue neurosis; occupation spasm.

Etiology.—This neurosis is more frequent in males than in females. It is very commonly found in writers, the condition being called *writer's cramp*. It is also found among telegraph operators, stenographers, violinists, pianists, shoemakers, tailors, cigar rollers, and compositors. It is worthy of note that writer's cramp is particularly apt to attack those

individuals who write a good hand. Neurotic temperaments, either hereditary or acquired, are very important predisposing factors. It may follow trauma or inflammation of certain parts of the body. Sexual excesses and the abuse of alcohol and tobacco predispose.

As yet no pathologic changes have been encountered.

Symptoms.—The commonest form of occupation neurosis is writer's cramp, which is here described; other varieties are similar and require no special description. The disease is very gradual in its onset. Writing becomes somewhat difficult, until finally spasm develops, which may be tonic or clonic, and prevents the proper performance of duties. The thumb and index-finger are most frequently involved. Sometimes the first three fingers and the hand may become locked during the act of writing or the pen be thrown out of the hand, or in other instances a tremor may develop, involving the hand and the forearm. In some cases weakness develops in the hand while writing, so that rest is necessary. Pain which shoots up the arm may accompany this muscular debility. Numbness and tingling of the hand and arm may occur in some cases, accompanied by severe pain. There may be vasomotor disturbances, the skin becoming pale and glossy or flushed.

Prognosis.—The prognosis is favorable only if the particular occupation be discontinued, for there is always a strong tendency to a return of the condition.

Treatment.—Rest is the most important measure. In some instances it becomes necessary for the patient either to give up writing or to learn to write with the left hand, but even in such instances a cramp may develop in the left hand, such cases having been recorded by Duchenne and Seeligmüller. Systematic massage, electricity, hydrotherapy, and certain drugs, such as iron, quinin, arsenic, cod-liver oil, the bromids, and hypodermics of strychnin and atropin, are useful.

HYSTERIA.

Definition.—Hysteria is a functional disease of the nervous system, characterized by a variety of symptoms which may simulate many diseases.

Etiology.—The disease is very much more common in women than in men. Heredity plays an important part in the causation of the affection. It is more frequent in the extremes

of the social scale, the poor and the rich being affected much more frequently than the middle classes. Injuries, such as railroad and other accidents, especially, according to the French observers, are the commonest causes of hysteria. It often follows the acute infectious fevers. Spoiled children are apt to become hysteric. Sometimes the disease shows itself in epidemics; especially is this true when it is due to religious excitement. It has been claimed recently that it may be due to alcohol, tobacco, mercury, and lead, and may result from sexual excesses and masturbation. Bad health is also a potent factor in the production of the disease, or it may result from overwork; this is particularly true in school-children. Grief, excitement, and disease of the genital organs, especially the ovary, give rise to hysteria.

Pathology.—No lesions have been found in this disease.

Symptoms.—The symptomatology of this disease is so varied and complex that a correct description is almost impossible. For this reason it has been found useful to give a brief description of a mild and a severe hysteric attack, and of the important symptoms which may occur either during or between hysteric manifestations.

Hysteric Paroxysm.—In the mild form the disease may begin either suddenly or after a period of malaise and nervousness, with disturbances and uneasiness in the epigastrium, also with palpitation and a sensation as if a ball were rising in the throat (*globus hystericus*), causing a feeling of strangulation, more rarely it is preceded by convulsive movements. There may be dizziness and vertigo, the patient weeping and falling upon a chair; there are breathlessness and crying, alternating with laughing. There are attacks of eructation of wind. The disease is frequently ushered in by spells of laughing or crying. The symptoms just enumerated are commonly encountered in the milder forms; in the severer ones the patient falls, always, however, selecting such a spot as not to be injured. Convulsions are common. Consciousness is never entirely lost, and the tongue is not bitten. Clonic and tonic spasms occur, but they show no regularity; the clonic may precede the tonic, and vice versa, and any group of muscles may be involved. These symptoms are accompanied by palpitation, tinnitus aurium, dimness of vision, and headache. As a rule, the temperature remains normal during the attack, or it may be slightly subnormal. The pupils are irregular; sometimes being dilated, or at other times contracted. Often during the

convulsions the body assumes an arched position (opisthotonos). It is common during the convulsive seizure for the patient to cry, laugh, scream, and sometimes talk incoherently. The attacks may assume any grade between the mildest and the severest forms, and may so closely simulate epilepsy that the physician who depends entirely upon the account of friends may for a long time be in doubt as to the diagnosis.

Sensory Phenomena.—Anesthesia, especially of the skin, is one of the most important phenomena. The senses of touch, of pain, and of temperature may be diminished or entirely abolished. In the severer cases there may even be anesthesia of the deeper parts. Hemianesthesia is common. Sometimes the anesthesia is distributed in different parts of the body with normal parts between; in rare instances the whole body may be affected.

Hyperesthesia and hyperalgesia are commonly encountered. Pain in the head is often severe, sometimes described as though a nail were driven through it; this has been called the *hysteric clavus*. Tenderness over the supra- and infra-orbital foramina is frequently noted, and pressure in this region may produce a hysteric attack (convulsion). The spine is often tender, particularly at certain points, and pressure in this region may also produce attacks. Similar areas of tenderness are noted in the inguinal region, especially upon the left side, in women as well as in men. Tender points are also met with on the limbs.

Motor Phenomena.—There is frequently inability to stand or walk; the muscular power, however, being retained while in the recumbent posture. The reflexes and electric reactions and the sensations are not impaired. A condition described as *astasia abasia* is characterized by the symptoms just enumerated, and is probably of hysteric origin; however, cases have been recorded which have been associated with chorea, epilepsy, and psychosis. Muscular wasting is usually not apparent in motor hysteric disturbances, but in rare instances wasting does occur, when it is general and not accompanied by fibrillary contractions. The wasting may come on and disappear rapidly. Various contractures are associated, which may be monoplegias, hemiplegias, or paraplegias. The patellar reflex is usually exaggerated. Babinski has described a reflex which normally is produced by gentle friction of the sole of the foot, causing the toes to become flexed, while if there be disease of the pyramidal tract, an extension of the toes,

especially the great toe, occurs. These signs are said to be always absent in hysteria.

Reflex Phenomena.—The reflexes are never absent in hysteria, and are usually exaggerated, in some instances being normal. The presence of well-developed ankle clonus is not a manifestation of hysteria, but a slight ankle clonus may be encountered in the disease. There may be paralysis of the sphincters, giving rise to retention of urine, and characterized by intermissions.

Tremors.—Several varieties occur; the commonest consist in rapid tremor of the hands, particularly noted during examination by the physician. There may also be tremor of the head and tongue. A coarse tremor is sometimes observed in the wrist and forearm, occasionally also affecting the legs.

Ocular Symptoms.—Ptosis may occur, and may be either unilateral or bilateral. There may be strabismus. Conjugate movement and diplopia are symptoms. Nystagmus is rare and the Argyll Robertson pupil is never noted in this disease. Photophobia is common, the patient preferring to be in a darkened room. There is frequently concentric narrowing of the visual field, and color sense is sometimes impaired and there is reversal of the fields. Some time after the hysteric attacks the visual field may return to its normal condition. Scotomata and hemianopsia are extremely rare; when present, organic disease should be suspected.

Auditory Symptoms.—Tinnitus aurium and vertigo are common. Occasionally hysteric deafness has occurred.

Aphonia.—This is common, the patient not being able to speak louder than a whisper. Mutism is rare.

Other Symptoms.—A harsh, unproductive cough is sometimes noted in this disease, and is known as the *hysteric cough*, there being no lesion of the larynx or lungs. The will-power is impaired. There may be noises that simulate cries of the lower animals. Even epidemics of this kind have been noted. Persistent hiccup, sneezing, yawning, dyspnea, and orthopnea without cyanosis have been observed. Dysphasia, eructations of wind, borborygmi, vomiting, nausea, and difficult swallowing are all symptoms. Tenderness of the joints, unaccompanied by inflammation, is present, the large joints being most frequently affected. Tenderness of the mammæ is not common; rarely upon palpation a tumor may be detected. Spurious tumors are sometimes encountered in

the abdomen, which disappear when the patient is under an anesthetic; these are known as *phantom tumors*. Pseudo-angina pectoris, palpitation, and vasomotor disturbances are common. In very rare instances hemorrhages are noted in the skin and from some of the mucous surfaces, so that hemoptysis and hematemesis may be encountered; however, this is an extremely rare condition and should be ascribed to hysteria only when local lesions can not be elicited and other hysteric manifestations are present. Ischemia and edema are also present in some cases. Mania, double consciousness, and hysteric insanity are rare manifestations.

Hysteric Fever.—Slight fever may be noted in hysteria. The temperature may be high for a considerable length of time or there may be paroxysmal variations, or it may vary in different portions of the body,—for example, being normal in one axilla and subnormal in the other,—but it is commonly normal. It is said that hysteric fever is not accompanied by the tissue changes that are so usual in the ordinary fevers, therefore the temperature sometimes reaches very high points; 110° F., 112° F., and even 118° F. have been recorded. The fever is frequently accompanied by abdominal and joint pains, so that under these conditions one must exclude organic lesions. Malingering should not be forgotten as a possible cause for apparent pyrexia, as not infrequently hospital patients are skilful in the handling of a thermometer.

Prognosis.—As a rule, the prognosis as to life is favorable. Death has been recorded by Charcot and Weir Mitchell, but this is extremely rare.

Treatment.—The physician must not be harsh in his treatment, but should always exercise tact, firmness, and kindness, and the patient should have faith in the medical adviser. The diet should be light but nutritious. Systematic exercise and sunshine are often of great value in the treatment. The rest-cure, as introduced by Weir Mitchell, is of benefit. Hydrotherapy and electricity are also indicated. Valerian, asafetida, and the bromids are of use. For the pain opium should not be employed; in fact, all narcotics are to be avoided, as there is great danger of acquring the drug habit. For the pain counterirritation is often of value, the thermocautery being applicable in some cases.

NEURASTHENIA.

Definition.—Neurasthenia is a functional disease of the nervous system characterized by mental and bodily weakness.

Etiology.—This is particularly a disease of centers of culture, affecting both sexes; the male sex, however, being slightly more often affected than the female. It is most frequent between the ages of twenty and fifty. Heredity plays an important part in this disease. Nervous parents often transmit their constitutional fault to their children; however, heredity is not always of prime importance in the causation of this disease. Worry, fatigue, excitement, and excessive mental or bodily strain are important causative factors. The most frequent causes among men have been unhappy marriages, financial anxiety, excessive study, and fear of venereal disease. Among women the causes have also been unhappy marriages, family cares, and dread of epidemic diseases, particularly cholera. The disease is particularly prevalent in the United States, although it is also frequent in parts of Europe. It is very common among the Jews. It is a disease of the well-to-do and of the educated classes. Of the acute infectious diseases, neurasthenia most frequently follows influenza, enteric fever, and malaria. Of the chronic infectious diseases, the most important as a causative factor is syphilis. The abuse of chloral hydrate, morphin, cocain, tobacco, tea, and coffee have been known to give rise to the disease. It may follow pregnancy or chronic local diseases, particularly chronic gastro-intestinal disease and diseases of the genitals. Floating kidney and dropping of other viscera have often been observed with this disease. Systematic attempts at curing obesity, sexual excesses, masturbation, and dietetic errors may give rise to neurasthenia. Various accidents, particularly railroad accidents, may cause the disease (traumatic neurasthenia).

Symptoms.—The symptomatology is very complex; indeed, so varied are its phenomena that special groups of cases have been differentiated, according to the predominance of certain symptoms. They are the *cerebral*, the *spinal*, the *gastro-intestinal*, the *cardiac*, and the *sexual* varieties. The general symptoms are despondency, fatigue upon slight exertion, both bodily and mental, and loss of weight; in some instances anemia, loss of sleep, and mental emotion are also frequent symptoms. There is particularly fear of being in a large open space (agoraphobia), and the fear of being shut up,

especially in crowded places (claustrophobia), these conditions being quite characteristic of the disease. The patients frequently relate to the physician their various symptoms, and often take note of them, reading the report to the medical adviser. This was called by Charcot "L' homme à petitis papiers." Hyperesthesia, vertigo, and depression are all common symptoms. The temperature is usually subnormal. The appetite is variable, and constipation is the rule. There is much irritability of temper.

The Cerebral Variety.—Headache, sleeplessness, inability to work, worry, depression, and anxiety are common symptoms in the cerebral type. There are very frequently ocular phenomena, reading tiring the patient. Sleeplessness is the rule, but in some instances the patient may sleep well. Disagreeable dreams are common. The patient is nearly always better in the evening and worse toward the morning hours. Flashes of heat and cold on the surface of the body, anemia, palpitation of the heart, and sweating are usual symptoms. Frequently there is a sensation of tenderness on pressure of the head.

The Spinal Variety.—Great weakness, especially upon rising in the morning, muscular stiffness, pain, and fatigue are common symptoms in spinal neurasthenia. It often requires many days for the patient to become rested, and fatigue again develops upon very slight exertion. Backache and tenderness at certain points along the spine are common symptoms. The deep reflexes are exaggerated. Ankle clonus is sometimes present. Paresthesia, especially formication, is a common complaint. There are sensations of heat, cold, and tingling in various parts of the body. Not infrequently symptoms of lightning pains are encountered, which in some degree simulate the pains of tabes; this pain, however, may be localized to distinct nerve-trunks, which are always tender upon pressure. Rarely Romberg's sign is present. Coordination may be somewhat disturbed, so that the acts of writing and talking are imperfectly performed. The nervous form of astasia abasia, as described under Hysteria, is present in this variety of neurasthenia.

The Gastro-intestinal Variety.—The gastro-intestinal variety is characterized by symptoms which particularly relate to the gastro-intestinal tract. Hyperacidity, pyrosis, nausea, vomiting, disturbed sleep, irritability of temper, sensations of flatness in the epigastrium, a bitter taste in the mouth in the

morning upon awakening, anorexia alternating with bulimia, constipation alternating with diarrhea, flatulency, occasionally tenesmus, and often rapid loss of weight are the common symptoms. Floating kidney and enteroptosis are frequently associated with the gastro-intestinal variety. Headache is a very common symptom.

The Cardiac Variety.—Palpitation of the heart upon slight exertion or slight excitement, precordial distress, pseudo-angina pectoris, and vertigo are symptoms of particular prominence. Arrhythmia and powerful pulsation of the arteries, particularly of the abdominal aorta, are common symptoms, these manifestations occurring without obvious changes in the walls of the blood-vessels. There is an absence of valvular disease. A capillary pulse may even be present in some instances. The extremities are frequently cold, and flashes of heat in the head are common.

The Sexual Variety.—This form of neurasthenia is characterized by spermatorrhea, nocturnal emissions, masturbation, perverted sexual desires, impotence, and painful testicle or ovary. Quacks flourish upon these poor individuals, their pamphlets and circulars being the net by which the victims are drawn into their traps.

Prognosis.—Under proper treatment the prognosis is favorable. It is well expressed by Allbutt in the following terms: "The patient who can lift his eyes to the future will recover; he whose thoughts live in the past is on the broad road to lunacy." The earlier the case comes under the observation of the physician, the more hopeful the prognosis.

Treatment.—Of all diseases, neurasthenia requires study of the individual case. The following methods of treatment have been very successful. The rest cure is applicable in many cases, while in others a change of climate is of great benefit. Hydrotherapy, electricity, massage, and hypnotism have all been of value in the treatment. The diet must be suited to the case, and must be easy of digestion. Milk diet is sometimes necessary. The bromids are of particular value in the cerebral variety. In gastric cases, arsenic in the form of Fowler's solution is of service. Syrup of the hypophosphites and valerian are also useful. Caffein, hyoscin, and phenacetin are of benefit. Opium and other hypnotics, if used at all, must be administered with great caution. Alcohol may be given sparingly. Iron and cod-liver oil may be used. Systematic exercise is of great benefit.

PART IX.

DISEASES OF THE MUSCLES.

MYOSITIS.

Definition.—Inflammation of the muscles.

Etiology. — This may be either primary or secondary. When primary, it is sometimes known as acute poliomyelitis. It may be due to trauma or to the invasion by parasites of the muscles, particularly by the trichinæ. Secondary inflammatory conditions of the muscles arise from acute and chronic diseases, in which there is either general or local parenchymatous or interstitial change, this being either suppurative or non-suppurative. Myositis often arises during the course of the specific fevers, such as enteric fever, typhus fever, and variola. Infection of the muscles takes place in the course of pyemia, ulcerative endocarditis, glanders, puerperal fever, actinomycosis, erysipelas, gonorrhea, and from some wounds. Finally, myositis is often due to syphilis.

ACUTE POLYMYOSITIS.

Definition.—Acute inflammation of any muscle group.

Etiology.—The disease is said to be due either to the influence of toxins or to an animal parasite. It has occurred in the course of pulmonary tuberculosis and diabetes. The disease is more common in males than in females, and does not occur in children.

Pathology.—Any of the muscles of the body may be implicated; the masseter and ocular muscles, however, commonly escape. The muscle-tissue is swollen and yellowish-white in color; occasionally there are brownish-red patches covering this decolorized area. Often hemorrhages may be noted. The muscle is soft and friable.

Symptoms.—The disease begins gradually with weakness, anorexia, headache, and occasionally vomiting, accompanied by subfebrile temperature without chills. These symptoms may be preceded by local pain, cramp-like in nature, with tenderness particularly in the arms or legs. The pain may become so severe that it is impossible for voluntary movements to take place in the muscle, but the joints are not affected. Soon muscles of other parts of the body become affected, such as the intercostals, the diaphragm, and the muscles of deglutition. Sensation is retained, the nerves not being tender upon pressure. The electric reactions of the muscle are normal. Stomatitis frequently occurs in the course of the disease. The spleen is commonly found enlarged. In the milder cases recovery takes place in the course of a few weeks; in the severer forms atrophy of the muscles may be found after the acute process has subsided, in which case the electric reactions are changed; and in the severest cases death may occur on account of the implication of the muscles of respiration, and bronchopneumonia is apt to arise.

Treatment.—Treatment should be directed to relief of the pain. In convalescence tonics are necessary.

MYOSITIS OSSIFICANS.

This is a rare disease. By many it is regarded as an inflammation, and by others as a new growth. It has followed slight injury. Ossification develops later, forming branching fragments, which may be either free or attached to the bone. The process may occur in the back, neck, and thorax, and gradually extend to all parts of the body. The entire muscular structure may become rigid. As a rule, this disease begins in early youth.

MYOTONIA CONGENITA.

Definition.—Thomsen's disease is characterized by prolonged contraction of the muscles concerned in voluntary movements when brought into action.

It is said that Thomsen's disease is the rarest in medicine. It was quite accurately described by Dr. Thomsen, a Danish physician, who suffered from the malady.

Synonym.—Thomsen's disease.

Etiology.—Heredity seems to play an important part. It

occurs most frequently about the age of twenty, and is more common in males than in females. A family history of neurotic temperament plays some part in the etiology.

Pathology.—The muscle-fibers have been found hypertrophied.

Symptoms.—The most important symptom of the condition is that upon voluntary movement the contraction of the muscle which the patient desires to move is slower than normal, and when contracted remains so for some seconds; this contraction may be so strong that the muscles which are apposed can not overcome it. This is well illustrated in the voluntary muscles of the hand. It requires some time for the patient to flex the fingers; the contraction of these muscles also being marked, it requiring ten or fifteen seconds to perform this act. The arm or the muscles of the face, those concerned in the moving of the jaw, the muscles of the thigh, the legs, the neck, and the back may all be affected. The muscles concerned in the acts of respiration, deglutition, micturition, defecation, and parturition are not affected. Sensory disturbances are not present, both the superficial and deep reflexes are normal.

Prognosis.—The prognosis is good as far as life is concerned, but the condition is incurable.

Treatment.—No treatment has any effect upon this disease. Thomsen thought that the more active his life, the less he suffered from the disease.

IDIOPATHIC MUSCULAR ATROPHY AND HYPERTROPHY.

Definition.—A disease characterized by atrophy and hypertrophy of various groups of muscles not associated with diseases of the anterior cornua of the spinal cord.

The disease appears to be congenital, although in many instances it does not show itself for some time after birth.

Synonyms.—Progressive muscular dystrophy; primary progressive myopathy.

Pathology.—The lesions are found in the muscles, the central nervous system not being affected. Interstitial change is noted in the muscles affected, there being either a deposit of fat or new-formed fibrous tissue. The muscle-fiber rarely reveals fatty degeneration or waxy degeneration. According to Erb, the primary changes are in the muscle-fiber itself.

Diagnosis.—The chief points in the diagnosis are the absence of the fibrillary contractions, the fact that sensation is not altered, and the absence of reaction of degeneration to electric testing. The onset is slow, affecting various groups of muscles.

Prognosis.—The prognosis is unfavorable as regards cure, although the disease may last for a number of years, the patient usually dying of some complication.

Treatment.—The most important measures are careful hygiene and the administration of tonics to sustain the general strength of the patient. Drugs seem to have no influence upon the disease. Massage may be employed and light exercise should be encouraged.

There are several varieties of the disease, all of them, however, characterized by lesions of the muscles without disease of the central nervous system.

Three varieties are described: (1) Pseudohypertrophic muscular paralysis. (2) The juvenile form of progressive muscular atrophy. (3) The facioscapulohumeral form.

I. PSEUDOHYPERTROPHIC MUSCULAR PARALYSIS.

Etiology.—It is most common in childhood, although the appearance of the disease may be delayed anywhere from childhood up to puberty. It is more frequent in the male than in the female sex, and heredity seems to play an important part. The disease is transmitted through the females, but principally attacks the males. It sometimes skips generations (atavism).

Symptoms.—An early symptom of the disease is difficulty in walking. The muscles apparently become very large, so that not infrequently friends seek the advice of the physician on account of this peculiarity. It is a peculiar fact that some muscles have a tendency to atrophy, while some have a tendency to become enlarged. The muscles of the calf are quite large. There is difficulty in attempting to stand on tiptoe, showing that the muscles have lost their power to some extent. The anterior tibials, the gluteus maximus, and the extensors of the thigh are also enlarged, while the flexors of the knee and hip-joint, also the adductors of the hip-joint, are atrophied.

The following muscles are hypertrophied: The deltoid (this showing the affection prominently), the supraspinatus and infra-

spinatus, the triceps, the extensors of the wrist and fingers, the muscles of the tongue, and the masseter. The following muscles are usually atrophied: The lower half of the pectoralis major, the latissimus dorsi, and the biceps. The following muscle groups are usually not affected: The trapezius, the rhomboideus, the serratus magnus, and the muscles of the hand, forearm, face, and neck.

There is difficulty in walking and in arising when in the sitting posture. The gait is peculiar, the patient throwing the knee in advance of the foot and throwing the shoulders from one side to the other. In the standing posture the feet are widely separated, so as to give the patient a better base of support. Contractures of the muscles sometimes occur early in the disease, which are due to the fibrous connective tissue in the muscles. The electric reactions are not altered at first, but later the electric contraction diminishes to the faradic and galvanic currents. Very late in the disease the reaction to the battery is entirely absent. The knee-jerk is at first normal, but it diminishes as the disease progresses. Fibrillary contraction is absent. Sensory disturbances are not encountered.

II. THE JUVENILE FORM OF PROGRESSIVE MUSCULAR ATROPHY.

This form differs from the first variety, just described, in that the disease occurs later in life, the muscles being wasted instead of being enlarged.

III. THE FACIOSCAPULOHUMERAL FORM.

This form is probably the same disease as the juvenile variety, differing only in that the muscles of the face are involved, and therefore they are described together.

Etiology.—Sex plays no part in the causation, both sexes being equally affected. There is a marked hereditary tendency. The disease is more common in childhood than in the very young or in adult life.

Symptoms.—In the juvenile form described by Erb the muscles of the shoulder and upper arm are first affected, revealing marked wasting and diminished function or paralysis, the muscles of the face usually escaping. Fibrillary contractions are absent and the reflexes disappear as the disease advances. In the facioscapulohumeral variety the atrophy most often begins in the muscles of the face. In this variety there

is often difficulty in closing the eyes. The orbicularis palpebrarum is atrophied, while the muscles of the eyeball usually escape. The orbicularis oris and the levator anguli oris are wasted. Many of the other muscles of the face are also affected in some cases. Most of the muscles of the shoulder-girdle and arm are also atrophied. Fibrillary contractions are absent, the electric reactions are slightly diminished, and the knee-jerks are normal until late in the disease. Sensory disturbances are not encountered. The course of the disease is prolonged.

PART X.

INTOXICATIONS AND SUNSTROKE.

POISONING BY FOOD; PTOMAIN-POISONING.

Food may act as a poison in one of two ways—either as a result of bacteria which have gained access to the food and produced their ptomains, or through an admixture of chemic poisons either organic or inorganic. The form of food-poisoning from chemicals of an organic or inorganic variety belongs to the domain of toxicology.

Food-poisoning due to bacteria may occur in one of three ways: First, the food may have undergone putrefactive changes, with the formation of toxic chemic substances (ptomains), before having been eaten, in which case the symptoms are extremely rapid in their onset. Second, the food may contain pathogenic micro-organisms, which, if they have been swallowed, set up toxic phenomena; in most instances of this sort there is a period of incubation, and this often precedes local lesions elsewhere than in the intestinal tract. Third, putrefaction may occur in the large or small intestine, the food having been in apparently good condition when swallowed.

Various ptomains, which were first studied by Selmi and later by Brieger, have been found in the cadaver. *Cadaverin*, *putrescin*, and *cholin* are only slightly poisonous. A muscarin-like alkaloid that occurs in decomposing flesh when swallowed gives rise to profuse diarrhea, lacrimation, salivation, and sweating, in which clonic spasms occur, with heart-failure.

Sepsin, obtained from decomposing yeast, when introduced into the system, causes vomiting and bloody diarrhea. Mydalein, derived from the human cadaver, causes a rise in temperature with dilatation of the pupil, paralysis, and convulsions. None of the other ptomains, as a rule, produces fever.

When fever occurs, it is due to the albumoses or digested proteids.

Symptoms of Food-poisoning.—The symptoms produced may be due to the bacterium itself, to its chemic products, or to a combination of both. If the symptoms be due to the bacterium itself, a period of incubation occurs that varies as regards the individual micro-organism. The onset of the disease is sudden. The patient is frequently seized with chill, abdominal pain, and muscular weakness. Vomiting and vertigo are frequent symptoms. Diarrhea with offensive stools, which may be bloody, accompanied by great thirst, is a prominent and almost invariable symptom. Some febrile reaction almost always occurs, the temperature in individual cases, however, showing great fluctuation. The pulse is rapid; there are muscular twitching, disturbance of vision, with dilatation of the pupils, and drowsiness. Urticarial and erythematous rashes show themselves. If death takes place, it is preceded by coma. Convalescence is often protracted.

Treatment.—A brisk purgative should be administered: 1 ounce of castor oil or from 3 to 5 grains of calomel, as it is necessary to rid the intestinal tract of the poisonous material. Opium is necessary to relieve the pain; and when there is profuse diarrhea, with offensive stools, intestinal antiseptics, such as beta-naphthol and salol, are of value. Stimulants are necessary for the prostration and for the cardiac asthenia.

GRAIN-POISONING.

This is an extremely rare condition. The principal effects of the poison usually fall upon the brain and nervous system, especially upon the spinal cord.

Ergotism.—Etiology.—The disease is due to the microbe known as the claviceps purpurea. Predisposing factors are starvation and ill health.

Symptoms.—The symptomatology shows itself in one of two varieties—first, the *spasmodic* or *convulsive;* and, second, the *gangrenous* variety. The acute form of the disease is most common in children. The symptoms are giddiness, depression of spirits, formication, with clonic and tonic spasms, colicky pains, tympanites, precordial distress, violent vomiting with purging, stupor, occasionally a vesicular eruption, and in the rapid cases the symptoms frequently resemble those of Asiatic cholera. When convalescence follows, which is rare,

it is prolonged and tedious, and sequels, such as epilepsy and cataract, appear. The gangrenous variety may also set in acutely, although the destruction of the limbs requires a longer time. Pain penetrates the affected part, but in some rare instances pain is entirely absent. An erysipelatoid eruption may precede the lividity of the part, but this is unusual, the lividity passing into a darker color, the limbs soon becoming black. Generally the gangrene is of the dry variety. More than one part of the body may be affected, and one part after another and one organ after another may be involved, until death takes place. If the disease be arrested, the patient may recover with the loss of a hand, foot, or limb. In some other cases the disease is more chronic, and in others there is a mixture of the spasmodic and gangrenous forms.

Diagnosis.—If the condition be epidemic, there is no difficulty in the recognition, but sporadic cases give rise to great difficulty in diagnosis. It has been claimed by Ehlers that Raynaud's disease, acrodynia, and erythromelalgia are only varieties of ergotism.

Prognosis.—The mortality in severe epidemics is 60%, and during less severe epidemics it may be about 10%.

Treatment.—There is no specific remedy, the treatment being entirely symptomatic.

Pellagra has already been described. (See p. 683.)

Lathyrism.—This is a name given by Cantani to an affection produced by diseased grain. The disease has been noted in parts of France, Italy, and Egypt. It follows an almost exclusive diet of chick-pea, with unsanitary conditions of life. These appear to be the most prominent predisposing causes.

Symptoms.—The symptoms come on rapidly, the patient being unable to arise from his bed in the morning, previously being in an apparently healthy condition. There is a stiff and creepy sensation in the limbs, and often a pain in the back is characteristic. Soon an unsteadiness in the hands, associated with tremor, occurs upon rising, and a peculiar gait is noted, resembling spastic paraplegia. Occasionally the gait may have an ataxic character, and shooting pains and great disturbance of sensation occur, such as hyperesthesia, paresthesia, and anesthesia. The tendon reflexes, however, are exaggerated. These symptoms require from four to five weeks to attain their full development. As a rule, the sphincters are not affected.

Prognosis.—The prognosis is generally favorable. If the

diet be changed soon enough before marked alterations take place in the cord, recovery may result.

Treatment.—The treatment consists in removing the cause, in putting the patient amid the best sanitary surroundings, and in administering tonics and stimulants.

MUSHROOM-POISONING.

This results from the eating of certain fungi which do not belong to the edible variety of mushroom, the toxic agent being muscarin, which is similar to those substances known as ptomains. Cholin is a substance closely allied to muscarin, and may also produce the same toxic effects.

Symptoms.—Shortly after a meal of poisonous mushrooms vomiting, diarrhea, severe cramps, and intense prostration promptly develop. Some disturbance of vision is common, muscarin having a myotic effect. Salivation is frequent, but perspiration is arrested. Muscarin paralyzes the cardiac muscle. In children convulsions are frequent.

Diagnosis.—This depends upon the contraction of the pupils, and upon the appearance of the fungus in the vomited material or in the stools.

Prognosis.—Recovery frequently occurs unless an enormous proportion of poisonous mushroom be taken and vomiting and diarrhea do not take place. Collapse and heart failure are the principal causes of death.

Treatment.—The stomach should be emptied and stimulants promptly administered, preferably hypodermically. The physiologic antagonist to muscarin is atropin, hence a hypodermic injection of atropin is almost a specific. Other symptoms must be treated as they arise.

SNAKE POISON.

Venomous snakes exist in nearly all the temperate and tropical parts of the world.

The venom may enter the body in different ways. It may enter through the subcutaneous tissue by means of the bite, thus reaching the circulation by absorption by the blood-vessels. If introduced directly into a vein, the effects occur much more quickly. Venom taken by the mouth, with the exception of the cobra venom, does not seem capable of producing poisonous effects, providing there is no abrasion in the

mucous membrane of the alimentary canal. The venom is excreted particularly by the kidney and to a slight extent by the salivary glands of the reptile.

Symptoms of Snake-bite.— Cobra-bite. — Immediately after a bite there is a sensation of burning and stinging in the wound, which soon becomes red, tender, and swollen. In the course of a half-hour constitutional symptoms begin to manifest themselves, such as desire to sleep, a feeling of intoxication, and weakness in the legs, which increases until the patient is unable to stand. Paralysis of the tongue, with profuse salivation, and inability to speak or swallow soon appear. With these symptoms there are nausea and vomiting. The signs of paralysis rapidly spread. The breathing becomes slower and the cardiac action quicker. After a while convulsions may occur; this is not necessarily unfavorable. The breathing ceases and the heart stops. During all this time the pupil is contracted, but reacts to light. Hemorrhages may occur from the mucous surfaces. The urine never becomes albuminous. If the patient recovers, convalescence is rapid.

Rattlesnake.—After the inoculation of the poison, severe pain takes place in the wound, accompanied by great swelling and discoloration, often slight bleeding occurring from the wound. Constitutional symptoms may appear within fifteen minutes. There is marked prostration, staggering gait, nausea, vomiting, cold sweats, dilated pupil, quick and feeble pulse. In a condition of this sort the patient may die within twelve hours after receiving the bite of a rattlesnake. If recovery occur from the depression, the swelling and discoloration rapidly extend, and there is a rise of temperature. The face becomes puffy and there are marked prostration and rapid syncope. Labored respiration, quick and feeble pulse, and a clear mind are characteristic. Occasionally there is restlessness, which may be preceded by convulsions. Suppuration may take place in the wound, and even gangrene may form, to which the patient may succumb several weeks afterward. On the other hand, the swelling may become less, the pain may gradually ameliorate, and recovery may occur. As a rule, when recovery occurs, it is rapid and will result in a few hours, even after the patient has been apparently in a moribund condition.

Prognosis.—The prognosis depends upon whether treatment has been prompt, and whether the reptile has embedded

its fangs and discharged the full amount of its poison into the system. The cobra venom is perhaps the most fatal.

Treatment.—The treatment should be considered under three headings: (1) To lessen the absorption of the poison; (2) to counteract the effect of the poison upon the system; (3) to hasten excretion.

A ligature should be tightly applied above the wound. Free incision must then be made into the subcutaneous parts around the wound. An elastic bandage should be applied downward several times. The application of nitric acid or of the actual cautery has been advised by Fraser. If the point of inoculation be a finger or toe, amputation after the use of the ligature is justifiable in some cases. The ligature can scarcely be retained for more than half an hour, as it produces great pain. It may then be removed for a few minutes, and when circulation has established itself, it can be reapplied. This process can be performed several times. Sucking the wound is to be recommended; precaution should always be taken to ascertain whether the mucous membrane of the mouth is not injured. The remedies which have been advised to counteract the effect upon the general system are intravenous injections of antitoxin and hypodermic injections of strychnin. Small doses of alcohol as a stimulant are useful, but exceedingly large doses would in themselves prove harmful. Walking the patient about is also useless. Recently antivenomous serum has been introduced by Dr. Calmette, which is said to be exceedingly effective. To hasten the excretion of the venom diuretics may be used.

ACUTE ALCOHOLISM.

The action of alcohol upon the system varies greatly in different individuals, both as regards dosage and effect. Some persons are extremely susceptible to its effects, small amounts giving rise to toxic phenomena. Others require large amounts to produce the poisonous effects.

Etiology.—Among the predisposing causes are idiosyncrasies and susceptibility, which may be due to heredity. The hereditary taint is probably responsible to a great extent for the use of alcohol. In individuals descendants of alcoholics the offspring are apt to be neurotic. Occupation is also a predisposing cause of some importance, as those engaged in the sale of liquors, those exposed to the vicissitudes of

weather, as cab-drivers, soldiers, sailors, and so on. The inhabitants of cold or temperate climates are more addicted to the use of alcohol than those living in warm climates.

Among the exciting causes may be mentioned mental depression, as from deaths of relatives, loss of money, failing health, pain, and so on.

Symptoms.—If a large quantity of alcohol be partaken of at one time, death may take place rapidly; this is, however, rare. It more frequently occurs that collapse results after the ingestion of large quantities of alcohol. As absorption takes place, coma develops, which is due to the narcotic effect of the alcohol upon the brain. The unconsciousness is not always complete, as in some cases the patient may be aroused by the use of the battery or a slap in the face, and so on. Paralysis does not occur. Usually there is some cyanosis of the skin or there may be flushing. The respiration is stertorous and the pulse full. The breath has an alcoholic odor, but not too much importance should be placed upon this, as an epileptic or a person having an apoplectic stroke may have partaken of liquor previous to the coming on of the symptoms. Convulsions are extremely rare. The temperature, as a rule, is normal or subnormal, and the pupils are dilated. Prompt treatment is most often followed by recovery.

Treatment.—If coma occur from alcohol, the stomach should be promptly emptied by lavage. Attempts should be made to arouse the patient by the use of electricity; strong coffee should then be given, and the patient should be placed in a warm bed. Collapse should be treated by hot applications and hypodermics of strychnin. When recovery has taken place, the gastritis, which is certain to follow, will require treatment.

CHRONIC ALCOHOLISM.

This results from the long-continued use of alcohol, and especially affects the nervous and digestive systems. At first functional, and later organic changes appear in different organs.

Symptoms.—The onset is insidious, the symptoms being fatigue, unwillingness to work, and loss of energy. There is malaise, headache, general depression, mental depression, loss of sleep, and tremor of the hands, lips, and tongue. The tremor is at first controllable. As the condition advances these early symptoms become more manifest. The skin becomes

flabby, the face may show venous congestion, and acne rosacea may show itself upon or about the nose. Symptoms of gastric catarrh are present. There may be injection of the conjunctiva, and perspiration occurs upon slight exertion. The tongue is flabby and furred, the tremor being marked when it is protruded. Leukoplakia may occur, especially in males. The breath is fetid and has a peculiar odor. There is frequently great thirst. There is often disgust for food, especially in the morning, which is made worse by the early morning nausea and vomiting. In severe cases the retching may cause hematemesis. In beer drinkers gastrectasis frequently occurs. Insomnia is an early and almost constant symptom; and if the patient sleeps at all, he is disturbed by bad dreams and nightmares. Peripheral neuritis may develop, first appearing as tenderness in the legs, which may be succeeded by sensations of pain, by wasting, and by paralysis of the muscles, affecting principally the extensors of the feet, so that foot-drop and loss of knee-jerks may result. The will and the intellectual faculties are greatly impaired; there is marked perversion of the moral tendencies, leading to falsehoods and deceit. The resistance of the body becomes lessened, so that when drinkers are attacked by acute disease,—as, for instance, croupous pneumonia,—they readily succumb.

Prognosis.—The prognosis is unfavorable.

Treatment.—The treatment consists in endeavoring to remove the cause. Such patients are best treated in an institution. Sleeplessness should be controlled by sedatives, although great care should be taken in using such drugs as morphin and chloral. It is better to rely upon trional, the bromids, and the hypodermic use of hyoscin. The general nutrition of the body must be looked after. Proper hygiene,—such as exercise in the open air,—bathing, massage, and so on, are of value. Many drugs have been recommended as specifics, but no one drug or method has been found successful.

DELIRIUM TREMENS.

Synonyms.—Alcoholic delirium; mania-a-potu; the horrors.

This may follow a single attack of hard drinking, but it occurs much more commonly in chronic alcoholism. It is most liable to affect persons who drink constantly without ever suffering from acute intoxication. Hereditary influences,

such as insanity, various neuroses, and alcoholism predispose to delirium tremens. The attack may occur in drinkers after trauma, after shock, or during the course of an acute infectious disease, such as croupous pneumonia. It occurs after surgical operations in alcoholics.

Symptoms.—The prodromes consist of nervousness, restlessness, and anorexia. There is insomnia; or if sleep occur, it is disturbed by bad dreams. The onset of the attack is marked by an aggravation of the prodromal symptoms. A tremor especially affects the lips, tongue, and limbs. Delirium soon develops, which is active and constantly changing. The patient experiences a sensation of uneasiness and has a desire to be in continual motion. The skin is wet, being bathed in perspiration. The expression is anxious. The pupils are dilated. The excitement is caused by fear due to the delusions and by aural and visual hallucinations; these are very frequently terrifying, the patient imagining he sees or hears serpents, insects, and dragons, which seem to come toward him. He may cease to recognize his attendants, and becomes suspicious; and while laboring under a hallucination he may attack his attendants. Frequently the patient may be controlled and humored by those understanding the condition. The temperature is subfebrile, rarely going above 103° F. The pulse is soft and rapid. The tongue is covered by a thick fur. There is complete insomnia. The disease is self-limited, lasting from two to four days. At the end of the period the patient falls into a quiet and peaceful sleep, and upon awakening is free from delerium; the tremor, however, persists for some time. In favorable cases convalescence is rapid. If there have been several previous attacks, hyperpyrexia may occur, which may prove fatal, or the patient may die from some complicating disease, particularly croupous pneumonia.

Prognosis.—In the first attack the prognosis is generally favorable. Old age, previous attacks, and complications are unfavorable prognostic signs.

Treatment.—Probably the most important feature of the treatment is careful feeding. The patient, as a rule, is half starved, and will show great unwillingness and reluctance to taking food. If necessary, a nasal tube should be used to administer food. For this purpose concentrated foods are best. But little food should be given at a time, but it should be frequently administered. Vomiting should be relieved by small pieces of ice and small quantities of effervescent mineral waters.

If vomiting be persistent, rectal alimentation must be resorted to. If there are signs of heart failure, stimulants must be administered. Strychnin hypodermically is of great value. A purge by calomel at the onset is often of benefit. Every effort should be made to induce sleep; the most useful agents are morphin hypodermically or hyoscin. Choral is also useful, but it has a depressing effect upon the heart. Complications must be treated as they arise.

METAL POISONING.

PHOSPHORUS.

Poisoning occurs in workmen occupied in the manufacture of matches; phosphorus is rarely used for suicidal purposes. Poisoning may be either acute or chronic; the chronic form is especially met with in those occupied in the handling of phosphorus for manufacturing purposes.

Symptoms.—Symptoms from a toxic process occur in from one to six hours after the drug has been swallowed. The poison has a disagreeable taste and smell, and pain soon develops in the region of the esophagus and stomach, this being followed by vomiting. The mucous membrane of the mouth and throat is dry and swollen. The vomited matter is often tinged with blood, and may be luminous in the dark and have the characteristic odor of phosphorus. The vomiting continues for several days, accompanied by diarrhea, insomnia, and great pain in the esophagus. The pulse and temperature are usually normal. About the third or fifth day jaundice appears, accompanied with the vomiting of coffee-ground material. The pulse-rate becomes slower, and, if the case be unfavorable, the pulse-rate increases; tachycardia may be a symptom. If fever sets in, it is an unfavorable sign. Marked changes take place in the liver, which is at first enlarged, but rapidly becomes atrophied. The urine is scanty, albuminous, loaded with biliary salts, and often contains crystals of leucin and tyrosin. Delirium and convulsions may now occur, giving place to coma. A fatal issue takes place in about 40% of the cases of acute phosphorus-poisoning.

Treatment.—Efforts should be made to prevent the absorption of the toxic agent, emetics and purgatives being

promptly administered. Sulphate of copper may be given as an emetic and lavage should be constantly used until the washings no longer have the odor of phosphorus. No fatty food should be given, not even milk or eggs, as fat dissolves phosphorus. Potassium permanganate, well diluted, is a good oxidizing agent, and should be administered early. The usual antidote is oil of turpentine. The bowels should be moved by salines or by enemas.

CHRONIC PHOSPHORUS-POISONING.

This occurs particularly in those engaged in the manufacture of matches, and the condition has been termed *phosphorism*. There soon develop a cachexia, showing a yellowish tint of the skin, the exhalation of a garlicky odor by the breath, with marked anemia, the occurrence of cystitis, bronchitis, and a necrosis of the jaw-bone, and the presence of phosphorus in the urine and saliva.

Treatment.—In the treatment of this condition a milk diet should be given. Moderate exercise, inhalation of oxygen, and repeated small doses of turpentine are of value. If a periostitis of the jaw-bone occurs, free incision with thorough drainage should be practised.

COAL-GAS AND WATER-GAS POISONING.

Illuminating gas owes its poisonous properties to carbon monoxid, which it contains from 5% to 10% of its amount. In water-gas the amount possessed reaches from 30% to 40%. This gas is often used for suicidal purposes, and the toxic effects vary with the amount of carbonic oxid inhaled. At first there may be only general feelings of discomfort, with throbbing of the vessels, marked headache, vertigo, and great debility, which is soon followed by nausea and vomiting. Drowsy sensations rapidly ensue, which terminate in coma, occasionally with delirium and convulsions. The skin becomes dusky, the lips and extremities are cyanotic, the pulse is full and bounding, and the respirations are labored. The patient dies asphyxiated. If convalescence occur, pulmonary and nervous complications may appear.

Treatment.—The patient should be removed from the influence of the poison as quickly as possible, artificial respiration being resorted to. Inhalations of oxygen and hypodermics

of strychnin are useful, but in all grave cases venesection and transfusion should be resorted to.

CHRONIC LEAD-POISONING.

Synonym.—Plumbism.

Etiology.—This condition occurs most often in workmen who are exposed in their occupations, by frequently or constantly coming in contact with lead or its compounds. It may, however, develop from the accidental absorption, frequently repeated, of small quantities of the metal. Lead may reach the system by the digestive tract or may be absorbed through the respiratory tract, skin, or mucous membranes. The compounds of lead, such as its salts or its alloys, may produce toxic phenomena; especially is this true of the form known as white lead, in which case the symptoms come on rapidly and are characterized by marked severity. Men are more frequently affected than women on account of greater liability to exposure. Children appear to be more susceptible than adults. Painters are frequently affected by lead-poisoning, as are also glass grinders, glaziers, and workmen engaged in the filling of storage batteries. Lead-poisoning may occur from drinking water which has become contaminated by flowing through leaden pipes or by being stored in cisterns. It may occur from biting colored threads which are stained with lead. Occasionally it is impossible to determine the source of the poison.

Pathology.—Changes are found in the peripheral nerves. The nerve-endings show a neuritis; even in the nerve-trunks degeneration may occur in patches, which is, however, more commonly marked toward the periphery, lessening in extent in the neighborhood of the cord. From this cause the muscles undergo atrophy. In cases of lead encephalopathy the vessels of the brain show arteriosclerotic changes, which may lead to softening, and hemorrhages following. Postmortem, lead has been found in the liver, kidneys, brain, and bone-marrow. The kidneys show the changes common to interstitial nephritis. The liver may show a similar change. It is probable that parenchymatous alterations precede the interstitial changes in the kidney.

Symptoms.—The symptoms presented by lead-poisoning show varying grades. As a rule, there are disturbance of nutrition, loss of flesh, anemia, arthritic pains, headache, and

general malaise. An important symptom is the blue line upon the gums, which is situated at the junction of the gum with the teeth in both the upper and the lower jaws. Occasionally the line is of a deep blue color. If the teeth are clean and free from tartar, the line may be entirely absent, or it may be present for a long time without marked symptoms of plumbism. It is due to the deposit of the sulphate of lead, which is formed by the combination of lead circulating in the blood with sulphureted hydrogen due to the decomposition of the tartar upon the teeth; thus, the line may appear very rapidly. It has been seen within twenty-four hours in persons after having used salts of lead for medicinal purposes. With this blue line the gums are frequently swollen and spongy. The breath is fetid. There is a metallic taste in the mouth and the tongue shows marked coating. Some grade of anemia is always present, although in the majority of cases it is of but moderate extent and of the chlorotic type. The erythrocytes are rarely less than 3,000,000; the hemoglobin, however, may be considerably diminished. The gastro-intestinal symptoms are of great importance. *Lead colic* is one of the characteristic phenomena of the affection. There is violent pain in the abdomen, which either may begin suddenly or may be preceded by slight pains for a day or two. There is marked tenesmus both of the bladder and rectum. There may be retention of urine, and even strangury. Obstinate constipation is the rule. Very rarely diarrhea is present. Often there are nausea and marked vomiting. When the vomiting is long continued, it has a biliary character. In lead colic the pulse becomes full and bounding; it is the high tension pulse. The rapidity of the pulse is lessened. Bradycardia may occur, and the pulse may be as low as 30 or 40 a minute; it is, however, regular in rhythm. As a rule, the respirations are increased in frequency. The amount of urine is diminished, and the entire attack may last a week. Relapses are very common, especially upon renewed exposure. Paralysis is a common symptom, affecting particularly the extensor muscles of the forearm, producing wrist-drop. As a rule, both arms are affected, although one may show the condition to a greater extent than the other. The paralysis often comes on suddenly, becoming well marked in from two days to a week. Other muscles besides the extensors may be affected. Occasionally the deltoid, the biceps, and the brachialis anticus are affected, but most commonly the biceps and the pectoral mus-

cles are implicated. When the small muscles of the hand become affected, atrophy of the thenar and hypothenar eminences appear most markedly, these being the earliest muscle groups involved. Paralysis of the legs is rare. Tactile sense is at first not implicated. Occasionally there is slightly diminished tactile sense, with some numbness in the affected muscles; however, pains in the joints are common. This condition has been called *saturnine arthralgia*. Blindness and convulsions are sometimes symptoms.

LEAD ENCEPHALOPATHY.

This condition is not common in lead-poisoning and occurs only in those who have become poisoned by large quantities of lead.

The symptoms consist in convulsions, delirium, and coma. As a rule, it takes from several months to a year to develop these symptoms, and a preceding history of colic, paralysis, and arthralgia is often obtained. Marked anemia and cachexia develop with vomiting and headache. There may be disturbance of vision, vertigo, tremor, insomnia, restlessness, mental depression, and more rarely hallucinations and delusions. Convulsions are the most common of the nervous symptoms which develop in severe cases. As a rule, there are repeated seizures, and they are characterized by the development of coma in the interval, in which the Cheyne-Stokes respiration may occur. This may be followed by the development of edema of the lungs, with hyperpyrexia, which may precede the fatal issue.

In the forms characterized by nervous phenomena the blue line upon the gums is a most important diagnostic symptom. Ocular phenomena occur, the most common being neuroretinitis. Primary optic atrophy, which may be advanced to complete blindness, has also been noticed in some cases. Young females affected by lead-poisoning are apt to develop amenorrhea and menorrhagia, and pregnant women commonly abort. The offspring of such parents are apt to suffer from convulsions early in life, which in the majority of cases are fatal.

Complications. — The common complications are those relating to the heart and kidneys. Hypertrophy of the left ventricle and interstitial nephritis develop in protracted cases. Gout has also been observed as a complication.

Diagnosis.—The diagnosis depends largely upon the history of exposure, the blue line upon the gums, lead colic, and paralysis of the extensors.

Prognosis.—Recovery often takes place from the most severe forms of lead-poisoning. Development of cerebral symptoms adds considerably to the gravity of the case. Complications relating to the heart and kidneys are permanent and progressive.

Treatment.—Efforts should be made to remove the patient from the influence of the toxic agent. The elimination of lead takes place through the urine and the feces. Lemonade, to which a few drops of sulphuric acid are added, followed by a dose of sulphate of magnesium may be found efficient in many cases. The usual treatment consists in the administration of iodid of potassium. For the lead colic opium in some form, preferably morphin hypodermically, is advocated. Constipation may be relieved by enemas of soap and turpentine. Atropin combined with morphin is of use in relaxing the intestinal spasm. When general paralysis occurs, strychnin is a valuable drug. Galvanism should be employed to keep up the nutrition of the muscles, and for the anemia iron in some form is the most useful drug.

CHRONIC MERCURIAL POISONING.

Synonym.—Ptyalism.

Etiology.—This condition has become comparatively rare owing to improved hygiene among the working-people. The disease is met with in quicksilver mines where the ore is heated. Mirror gilders, thermometer makers, hatters, and furriers who use the salts of mercury are apt to manifest the toxic effects of mercury. It sometimes arises from the internal administration of mercury. Some persons show a marked tendency or susceptibility to the drug, even to minute doses of calomel.

Pathology.—In cases of acute poisoning an intense inflammation of the gastro-intestinal tract is produced. Acute inflammation of the kidney is liable to occur in these cases. The peripheral nerves are sometimes affected, owing to the degeneration of the sheath, whereas the axis-cylinders remain intact.

Symptoms.—The symptoms are those of mercurial stomatitis described in the previous section. (See p. 437.)

CHRONIC ARSENICAL POISONING.

This may arise from the inhalation of arsenic in wall-paper, through certain molds which have the power of liberating arsenic in the form of a vapor, this condition being favored by moisture; hence it is liable to occur in damp weather. Arsenic is frequently present in the coloring-matter of various artificial flowers and hangings, dark-red cotton papers, labels, papers used in kindergartens, etc. It is used by the taxidermist in stuffing birds and various animals. From the internal administration of arsenic slight toxic effects are produced, and cases of peripheral neuritis due to this cause have been described.

Pathology.—The peripheral nerves show degenerations similar to those occurring from lead. Fatty degeneration of the liver-cells has been noted with infiltration of leukocytes.

Symptoms.—In chronic poisoning from arsenic catarrhal symptoms, particularly of the gastro-intestinal tract, occur. Symptoms referable to the nervous system and skin are also prominent. The most characteristic of these symptoms are those of the nervous system, and are due to the extensive neuritis, closely resembling that form due to alcohol and lead, which, as a rule, begins in the lower extremities with sensory phenomena. There are numbness and tingling, and hyperesthesia, paresthesia, and later anesthesia in the nerve-trunks and muscles. Paralysis occurs, particularly affecting the lower extremity. There is loss of knee-jerk, which is an early and almost constant symptom. Rapid atrophy occurs in the muscles, which present reactions of degeneration. As a rule, the sphincters of the bladder and rectum are not affected. Among the gastro-intestinal symptoms are nausea, vomiting, coated tongue, with a metallic taste in the mouth, and severe epigastric pain. Coryza occasionally occurs, and hematuria has been noted in some cases. Various eruptions upon the face occur, particularly urticaria, and herpes zoster has also been noted. Pigmentation of the skin is common and persistent. Anemia of the chlorotic type occurs.

Prognosis.—As a rule, even the severe forms of paralysis are amenable to treatment, although the affection is a chronic one.

Treatment.—The cause should, if possible, be removed. Iodid of potassium is indicated. Neuritis should be treated as in other forms of poisoning, by electricity, massage, strychnin, and tonics.

CHRONIC SILVER POISONING.

Synonym.—Argyria.

This condition is most frequently due to the long-continued use of the silver salts as a drug. Silver is deposited in the tissues, where it is recognized under the microscope as small black particles of metallic silver. This is best seen in the skin and in the kidneys. The discoloration of the skin is marked, often of a deep blue color, later not unlike intense cyanosis. The mucous membrane and the teeth are also discolored. A black line is often present on the gums. Parenchymatous changes occur in the liver and kidneys. (See p. 32.)

SUNSTROKE.

Definition.—Sunstroke is a disease characterized by prostration, with high fever, followed by coma; and is due to exposure to the rays of the sun.

Synonyms.—Heat stroke; thermic fever.

Etiology.—The disease occurs most frequently in hot climates in which the humidity is high. Alcoholism is a marked predisposing cause. It results from exposure and exertion in the sun. Elderly persons with chronic ailments, are liable to have an attack of sunstroke. Absence of drinking-water is said to be a predisposing cause with soldiers upon the march. The white race is more liable to be affected than the negro race.

Pathology.—The brain and its membranes, the lungs, and some of the abdominal viscera often reveal marked congestion. The vena cava and the right auricle are often distended with partially coagulated blood, which is dark red in color, almost black. Granular degeneration of the ganglion cells of the brain and spinal cord has been observed, and in some way seems to have a relationship to the symptoms. Parenchymatous degeneration is also observed in the liver and kidneys. Polycythemia may exist, which is probably due to blood concentration. If recovery occur, anemia develops, showing probably that hemolysis has been pronounced. The leukocytes are increased in number, and some of them may contain blood-pigments.

Symptoms.—As a rule, prodromes occur, which may be present for a few hours or several days before the attack

shows itself. On the other hand, the attack may begin suddenly. The premonitory symptoms usually are restlessness, sleeplessness, rapid shallow breathing, precordial distress, nausea, and sometimes vomiting, thirst, frequent micturition, and some rise in temperature. These symptoms gradually increase in severity, and the temperature reaches from 106° F. to 115° F., and is often higher. Dyspnea is marked, and the skin of the entire body is red and sometimes livid; it is most often dry, but occasionally some moisture is noted. The pulse is full and bounding and the pupils are contracted. Cheyne-Stokes respiration is noted as unconsciousness passes into complete coma. Convulsions may occur, and in individual cases the pupils may dilate. There is relaxation of the sphincters preceding death.

HEAT EXHAUSTION.

This is a condition characterized by prostration and normal or subnormal temperature, due to exposure to excessive heat. It is commonly met with in the working-classes, as firemen of ocean steamers, bakers, and laundry workers. The disease is commonly encountered in alcoholics, also in individuals who wear unsuitable clothing. It is more frequent among males than females.

Symptoms.—In heat exhaustion, as a rule, premonitory symptoms occur which consist in uneasiness in the epigastrium, dizziness, headache, irregular pains in the extremities, and occasionally tingling. Often there are nausea and vomiting. The prostrationg radually increases and the headache becomes marked. The patient appears dazed, and sensations are but partially perceived. The respirations are slightly increased, however; the pulse becomes rapid,—from 130 to 140; the temperature may be normal or subnormal; occasionally some slight fever is noted. As a rule, the patients recover.

Complications.—Insanity and meningitis may be complications. Occasionally pneumonia develops, and persons with renal disease develop uremic attacks. Cerebral hemorrhage has been known to follow sunstroke. The sequels consist in headache which may last for a long time, vertigo, general muscular soreness, and tingling in the extremities, this sometimes lasting for weeks or months. Anemia, as a rule, is marked. Epilepsy has followed cases of sunstroke. Persons

who have had an attack are extremely liable to second attacks upon exposure.

Prognosis.—In those cases of heat exhaustion that come under treatment early the prognosis is good. In sunstroke when the temperature reaches 110° F. or 112° F. and over, if properly treated, about half of the cases recover. The prognosis is less favorable in alcoholics and in persons subject to chronic disease of the heart, lungs, or kidneys.

Treatment.—The treatment of thermic fever consists in the application of cold to the surface of the body, preferably in the form of an ice-bath or ice rubbing. The cold pack is sometimes substituted. The bath is commonly continued until the temperature reaches the normal point. Ice-water enemas may also be employed.

In heat exhaustion the patient should be placed in a warm bath, and stimulants, such as ammonia, strychnin, whisky, ether, and nitroglycerin, administered. For the anemia that follows iron and arsenic are indicated. Antipyretics, such as antipyrin, may be employed, particularly in those cases in which a cold bath can not be given.

PART XI.

DISEASES DUE TO ANIMAL PARASITES.

PROTOZOA.

The amœba coli belongs to the order of protozoa. It varies between 20 and 30 μ in diameter. The organism consists of hyaline protoplasm, the cell-wall not being discernible. In the protoplasm there are vacuoles which may vary in size. The protoplasm occasionally contains foreign matter, such as bacteria, starch globules, and blood-cells. It possesses contractility, pseudopods being thrown out when the organism is in motion; therefore, the parasite is globular, oval, or irregular.

The parasite is best studied by taking a specimen from a stool that has just been passed, and the examination had better be conducted upon a warm stage. It is most often found in masses of bloody mucus. The amœba coli stains faintly with the basic aniline dyes, and can be well studied in the stained specimens when treated with toluidin-blue (as recommended by Harris).

The discovery of this parasite was made by Lösch, in the year 1874, in St. Petersburg. The amœba coli is associated with tropical dysentery; it is found in the stools, in the coats of the bowel, also in tropical abscesses of the liver, and rarely in other parts. A few observers believe that it does not possess pathogenic properties. (For description of Amebic Dysentery, see p. 298.)

Hematozoa of Malaria.—(See p. 198.)

Psorospermosis.—Psorosperms, or coccidia, which are frequent in the lower animals, occasionally occur in the human subject. When affecting man, the disease is at first local, later it shows a tendency to invade the system, becoming diffused

in different parts of the body, and sometimes causing death. The psorosperm attacks particularly the epithelial cells of the mucous membranes and of the skin, extending into the surrounding tissue, in this manner entering the lymph- or blood-vessels.

Pathology.—The disease produced by the *coccidium oviforme* has been particularly studied in the rabbit. It affects the liver especially, numerous minute firm nodules being found in the organ. The nodules vary in size, and may attain a diameter of about ⅓ of an inch. They contain a central, caseous mass, which can readily be expressed or separated. The coccidia measure from 26 to 28 μ in length, and from 13 to 14 μ in breadth. They are found in three stages, according to the degree of development. In the least mature the contents are generally granular. A further stage of development shows the granular material gathered to a central spheric mass, and in the fully developed form the granular mass divides into four psorosperms or spores. These are ready to undergo further development under suitable conditions. The parasite belongs to the order protozoa; subdivision, coccidiidea.

The disease has been observed in man both as a local and as a general condition. Locally, it has been met with in the liver, producing conditions similar to those observed in the rabbit.

The **symptoms** observed are progressive wasting, nausea, and exhaustion, followed by a fatal issue.

If the parasite affects the urinary passages, increased frequency of micturition with hematuria takes place; small superficial cysts with clear contents are passed. It may block the urinary passages, and so cause hydronephrosis. It may attack the intestinal mucous membrane, giving rise to constant diarrhea, hemorrhage, exhaustion, and general dyspeptic symptoms.

As a general disease, there are few characteristic symptoms. There are pains in the limbs, headache, drowsiness, delirium, dry furred tongue, nausea, vomiting, albuminuria, fever of a remittent type (rarely above 103° F.), and increased size of the liver and spleen. Symptoms of the "typhoid state" intervene. Death takes place in from two to seven weeks.

Cercomonas and *trichomonas intestinalis* are found in the intestines and stools; they probably do not produce disease.

WORMS.

Worms are divisible into various classes; only two, however, occurring in man. These are (1) the *Platyelminthes*, or flat-worms, which have flat, elongated bodies; the heads are provided with hooklets or suckers or both, possessing a cerebral ganglion, and, as a rule, being hermaphroditic; (2) the *Nemathelminthes*, or round-worms, may be defined as having tubular bodies. The end representing the head is provided with hooks or papillæ; the sexes are distinct.

FLAT-WORMS.

(a) **Cestodes.**—The cestodes, or tapeworms, are long, ribbon-like, flat, whitish organisms, which when matured inhabit the alimentary canal of many of the vertebrate animals.

The worm may be divided into a head, a neck, and a number of segments. The head has from two to four discs, sometimes called suckers, and in some of the varieties these are arranged around a rostellum, and occasionally there are hooklets. These hooklets help to hold the parasite in the intestine of the host. The head is very small, and in the majority of cases can just be distinguished with the naked eye. It shows itself as a small bulbar point at the end of the narrowest point of the worm; this is joined to the series of segments known as the proglottides. Segments gradually increase in size until they become more mature. The matured proglottides possess male and female organs of reproduction, the latter containing the eggs. As the ova mature the proglottides containing them break off and pass out of the body of the host by their own movements or with the feces. In a few species the ovum at birth is undeveloped, but, as a rule, the embryo is covered by a shell. The embryo, in most instances, possesses about five or six hooklets. As it arrives in its intermediate host (ox, pig, fish, etc.), the shell in which it is contained is dissolved and it bores its way through the walls of the intestine and the intervening tissues until it finds lodgment in the muscles, liver, lungs, or some other tissue. The hooklets are now cast off and a head and neck, which conform to the type of the tapeworm in general, are developed. The scolex, or larval form is contained in a clear, watery bladder, which is called the cysticercus. In some forms of tapeworm this cyst is very small and rudimentary.

In another variety (tænia echinococcus) the cyst formation becomes extensive, and from a process of development of the inner cellular layer of the wall of the cyst a number of smaller capsules are formed. In the fourth variety there is no cyst; the embryo enlarges, the head and two grooves being formed at the end; these are known as the bothriocephalidæ.

Many worms retain their power of development for a long period, sometimes years; others soon die and become calcified.

The tapeworms, or cestodes, in man are represented by two families—the *tæniadæ* and the *bothriocephalidæ*. The tæniadæ have four suckers, and some have a single or double row of hooklets placed upon a rostellum; the bothriocephalidæ have only two suckers, as a rule.

In man but three species of fully developed tapeworm commonly occur—the tænia saginata or mediocanellata, or beef tapeworm; the tænia solium, or pork tapeworm; and the bothriocephalus latus, or fish tapeworm. Some other varieties are occasionally met with, but they are rare. The tænia echinococcus in its larval state occurs in man.

Tænia Saginata, or Beef Tapeworm.—The head is surmounted by four suckers, with a rudimentary sucker in its center. The segments measure from 8 to 10 mm. in breadth and about 18 mm. in length. At the sides of the proglottis are placed the genital pores. The length of the worm varies from 4 to 10 meters. The ova are oval and measure about 0.03 mm. in their longest diameter. Hooklets probably can not be seen in the embryo; however, some observers hold that they do exist and can be demonstrated.

Tænia solium, or **pork tapeworm,** is sometimes called the armed tapeworm because the rostellum is supplied with two rows of hooklets, each row containing from 12 to 14 hooklets. The head is quadrilateral, also being supplied with four suctorial discs. The worm measures about 4 meters in length. The segments measure from 6 to 8 mm. in breadth and from 10 to 12 mm. in length. Behind the middle of the proglottis are found the genital pores, the uterus being less branching than in the tænia saginata. The ova are about the size of those of the beef tapeworm. The embryo is supplied with hooklets.

Bothriocephalus Latus, or Tænia Lata.—The length of the worm varies from 5 to 16 meters. The head is elongated and supplied with two grooved suckers, one on each side. The breadth (1.8 cm.) of the joints is greater than the length. The mature segments show a rosette arrangement of the

uterus which is quite characteristic. The eggs are oval, measuring about 0.07 mm. in the longest diameter.

Symptoms Produced by Tapeworms.—The tapeworm may attain considerable size, and may infest the host for a long period of time without giving rise to symptoms. The diagnosis of tapeworm in such cases can be made positive only by finding segments. As a rule, the appetite is good; often there are colicky pains and alternating diarrhea and constipation. There may be dyspeptic symptoms with nausea, especially when the stomach is empty. There may be anemia, and, as a result of the presence of the bothriocephalus latus, extreme anemia has often been observed. Occasionally there are nervous symptoms, such as vertigo, chorea, and epileptic seizures. All symptoms are apt to become aggravated when the stomach is empty. It is said that the symptoms are more severe from the bothriocephalus latus on account of its larger size.

Treatment.—The most reliable anthelmintics are the male fern; the pomegranate root bark, and its alkaloid, pelletierin; kousso; pumpkin seeds; turpentine, and chloroform. Thymol is also highly recommended by some authorities. Filix mas is a very reliable vermifuge. Before the administration of any of these drugs the patient should be put upon a milk diet for at least twenty-four hours; it is then desirable the night before the vermifuge is administered to give a brisk cathartic, preferably calomel. Early the following morning the anthelmintic is administered, and a few hours afterward another purgative. It is always necessary to search for the head, and it must be remembered that if the head does not pass, the worm will rapidly reform. Occasionally two or more worms may infest the same host, but if no symptoms appear for three months after the worm has been passed, a cure may be considered to have been effected.

Tænia Echinococcus.—*Synonym.*—Hydatid worms.

It is a small parasite, from 4 to 5 mm. in length, and about 0.6 mm. in breadth, and is composed of five segments, the terminal one being the largest. It is an exceedingly rare parasite, and is found in the intestine of the wolf and the dog. The head is supplied with 4 suckers and about 24 hooklets, situated upon a rostellum in a double row. The ripe segment contains about 5000 eggs, which further develop in many organs, particularly in those of the hog and the ox, more rarely in the sheep and the horse. In some climates man may be the host from accidental ingestion of the ova.

The embryo has six hooklets, and when liberated from its shell, it works its way through the intestinal wall into the organs. Commonly it enters the portal vein, lodging in the liver; it then may find its way into the hepatic veins, passing to the lungs, and sometimes by way of the pulmonary vessels to the left side of the heart, and reaching other organs.

When it becomes embedded, a small cyst forms. The cyst contains a clear fluid and the cyst-wall consists of two layers —the external capsule, which is laminated, and the internal, granular or parenchymatous layer, known as the endocyst. Chronic inflammatory reaction appears in the surrounding tissues, and in a short time fibrous tissue forms.

In the growth of the cyst buds develop from the internal layer, which are also converted into cysts, being identical with the original one. These are called secondary or daughter, cysts. At first they are connected with the primary cyst, but later are set free. In this way, from the primary cyst a dozen or more daughter cysts may develop. A similar process goes on in the daughter cysts. From the lining membrane growths develop, known as scolices, which are actually the head of the tapeworm (the tænia echinococcus), presenting a circle of hooklets and four sucking discs. Each head when entering the intestine of the dog is capable of developing into an adult tapeworm.

The fluid of the echinococcus cyst is clear and limpid, and has a specific gravity of from 1005 to 1009. Traces of sugar are present, but no albumin. The fluid of the cyst contains the characteristic hooklets, except when degenerative changes have taken place.

The embryo form of the echinococcus may remain alive for many years. When death takes place, it occurs from a granular change or calcification of the cyst. If the cyst ruptures, perforation may take place internally or externally. Occasionally even recovery may follow rupture. Rarely suppuration appears, large abscesses having been found that were due to hydatids. Hydatid disease is common in Australia and in Iceland; it is rare in Europe and America. The most common seat of the cysts is in the liver, as about 45% of all the cysts are found in this organ; the next most frequent seat is in the lung or pleura.

Symptoms.—Only large cystic formations give rise to symptoms. The physical signs, of course, will depend upon the situation of the growth. They will be those of a tumor, at times

firm, rarely with fluctuation. Frequently a tumor may be noticed to the left of the suspensory ligament of the liver, pushing up the heart and giving quite an extensive area of dullness upon percussion. If the cyst be superficial, it may give rise to a condition known as *hydatid fremitus* or *hydatid thrill.* This is elicited by palpating lightly with the fingers of one hand, percussing at the same time with those of the other; a vibration or trembling in the mass being the result, which may last for some time. With suppuration, symptoms of pyemia will develop. Occasionally from the rupture of a hydatid cyst urticaria develops. This sometimes follows aspiration.

Diagnosis.—This depends chiefly upon the finding of the hooklets in the fluid from the cyst.

Treatment.—Operative measures should be resorted to if the cyst becomes large or gives rise to pressure symptoms. Simple aspiration is all that is necessary in quite a number of cases. Injections into the sac are useless.

(b) **Trematodes.**—These are flat, leaf-shaped, unsegmented worms, possessing a mouth and a pharynx. They are most often hermaphrodites. In some few species the sexes are separate.

The trematodes which occur in man belong to the family distomidæ.

Distomidæ.—Those that occur in man are oviparous. The ovum at birth contains a ciliated embryo. On leaving the uterus of its parent it is carried in the discharges of the host into water or muddy soil; here the embryo is hatched out, and for a short period swims around and searches for its special intermediate host. This is most often a mollusc or crustacean.

Several forms of distoma are found in man, the most common being those found in the liver, known as liver flukes; those occurring in the blood, known as blood flukes (bilharzia hæmatobia); and the form found in the bronchial tubes, known as the bronchial flukes, or ringeri. In the liver two or three varieties of the distoma have been found—the distoma hepaticum, which attains a length of from 28 to 32 mm.; the distoma lanceolatum, being much smaller, from 8 to 10 mm.

There is a certain distoma which is endemic in some parts of Japan. According to some authorities 20% of the inhabitants of certain provinces in Japan are affected by this parasite. The parasite occupies the upper portion of the small intestine and the biliary passages. When present in large numbers,

an exceedingly fatal disease of the liver is produced, accompanied by ascites and jaundice, in which the liver enlarges enormously.

The bilharzia hæmatobia is found in Southern Africa, Egypt, and parts of Arabia, and gives rise to hematuria. The parasite lives in the venous system, particularly in the portal vein, and in the veins of the bladder, spleen, and mesentery. It is still unknown how the parasite gains access to the body; most likely, however, from impure drinking-water.

The most important symptom is a marked hematuria, which rapidly produces anemia; pain usually being present during micturition. The urine contains the ova of the bilharzia, which is readily recognized under the microscope, being ovoid in shape and translucent. The majority of cases recover.

The important symptoms produced by bronchial flukes are hemoptysis and the presence of the parasite in the sputum.

ROUND-WORMS.

Ascaris Lumbricoides.—The ascaris lumbricoides is one of the most frequent human parasites, occurring particularly in children. It is cylindric, pointed at both ends, and has a yellowish-brown or reddish color. The male measures 250 mm. in length, and the female 400 mm. The ova, which commonly occur in the feces of the host, are small and elliptic, with a thick covering, measuring 0.075 mm. in length, and 0.058 mm. in width. They develop outside of the body, but their life history is unknown. The parasite infests the upper portion of the small intestine; usually very few are present, but occasionally enormous numbers may exist. They may pass into the stomach, from which they may be vomited. In some rare instances the bowel has been perforated and peritonitis has occurred.

Symptoms.—The symptoms are indefinite. The child is usually irritable and fretful, picks at the nose, and grinds its teeth. There may be twitchings and convulsions.

Treatment.—Santonin in from three-grain doses in the child to proportionately large doses for the adult, followed by calomel or a saline, is sufficient.

Oxyuris Vermicularis (Pin-worm, Thread-worm).—This common parasite infests the colon and rectum, producing great itching and irritation, especially at night. The female measures about 10 mm. in length, the male being smaller.

The parasite is most common in children, but occurs at all ages. The worm is easily detected in the feces.

Treatment.—The treatment consists in the use of santonin in small doses, with mild purges. Large injections containing vinegar, quassia, aloes, turpentine, or carbolic acid may be employed. These injections should be continued for at least two weeks.

The ankylostoma duodenale is the only parasite of this variety which is injurious to man. It belongs to the same variety as the strongylus armata which causes aneurysms in the horse. The parasites chiefly inhabit the small intestine. The ova measure about 0.05 mm. in their longest diameter. The male has a length of from 6 to 10 mm., and the female from 10 to 18 mm. The head is somewhat rounded and supplied with six tooth-like hooklets, by which the parasite fastens itself to the mucous membrane of the intestine. It occurs in Egypt, in Europe, in India, in Brazil, in Jamaica, and perhaps in the Southern United States.

Symptoms.—The principal symptoms are those of anemia, owing to the fact that the parasite withdraws the blood of the host. At first there may be only slight gastro-intestinal disturbance, but when the parasite increases in numbers, anemia is characteristic. Egyptian chlorosis is due to this cause. The anemia may develop acutely and reach a high grade in a short time, with dyspnea and edema.

Diagnosis.—The diagnosis can be made only from the presence of the parasite or of the ova. The larvæ develop in mud, and easily find their way into drinking-water, in which manner infection easily takes place.

Treatment.—Thymol and male fern destroy the parasite. Prophylaxis consists in thoroughly boiling and filtering the water used for drinking purposes.

Trichinæ, or **trichina spiralis,** when they reach their adult size, infest the small intestine, trichiniasis being produced by the embryos, which, in their passage from the intestines, reach the involuntary muscles, where they become encapsulated, and are then known as muscle trichinæ.

Trichinæ occur in two different forms in the human subject—in the muscle and in the intestine. The trichina is occasionally found in the feces. The male parasite measures 1.5 mm. in length, the female being 3 mm. The male has four prominent papillæ placed between the conical protuberances at the extremity. The eggs develop into embryos while still in the

uterus. The new-born parasite, perforating the gut, becomes embedded in the muscles of its host. The worm possesses a somewhat pointed head and a rounded tail.

The young produced by each individual trichina is estimated at several hundred. When the trichinæ reach the muscle, they penetrate the muscle-fibers, and here exist in their embryonic state. From this process an interstitial myositis occurs. The capsule develops about the parasite. Several worms may be present in a single capsule. This process has been estimated to take about six weeks. The parasite does not undergo any further development, the capsule becoming thicker and lime-salts infiltrating it.

In man this process requires from four to six months; in the hog it may take many years. The trichinæ may live in the muscle for an indefinite period. They have been found active after having resided in the muscle of man for from twenty to twenty-five years after their entrance into the system. Often the worms are completely encapsulated.

Trichinæ occur in hogs, rats, mice, cats, foxes, and other animals. Dogs are infected with great difficulty; cats, more readily. An animal in which the living trichinæ swarm may be well nourished and look perfectly healthy.

The mode of infection in man is through eating the flesh of trichinous hogs. Systematic examination of the meat is extremely important. In Germany, where a systematic microscopic examination is carried on, trichinous hogs are found in the proportion of 1 in 1852. In England and the United States systematic inspection is unknown. "Taking all the examinations of American pork thus far made, both at home and abroad, and we have a total of 298,782, in which trichinæ were found 6280 times, being 2.1 %, or 1 to 48" (Salmon, 1884). The disease often appears in epidemics, large numbers of persons being affected from the same source.

Symptoms.—The disease is not caused when only small portions of trichinous meat are partaken of; few embryos may find their way to the muscles, and thus no symptoms are produced. In well-marked cases a gastro-intestinal period occurs, followed by a period of general infection. After eating infected meat, in the course of a few days symptoms of gastro-intestinal disturbances develop, such as loss of appetite, nausea, vomiting, and occasionally diarrhea. These prodromes are by no means constant. In other cases the onset may be acute, with severe abdominal symptoms, often resembling an attack of cholera.

Some weakness and debility, and pains in different parts of the body, have been noticed. The symptoms of the constitutional disturbance take place in about ten days, sometimes a little later. As a rule, there is fever, which is absent only in the mildest cases. Chills are rare. The temperature is either remittent or intermittent, and may vary between 102° F. and 104° F. As the parasites attack the muscle-tissue, symptoms of myositis develop. There is marked pain upon movement and pressure of the affected parts. The muscles are swollen. Edema often occurs early and is well marked in the face; later, it appears in the extremities. There is marked sweating with urticaria. As a rule, consciousness is not disturbed, except in some cases of extreme gravity, in which the "typhoid state" develops. Symptoms of delirium with tremors, dry tongue, and tympanitic belly then occur. Often there is dyspnea and some bronchitis. Pneumonia and pleurisy may occur as complications. Polyuria has been observed as a symptom in some cases. As a rule, albuminuria occurs, and the blood shows a marked increase in the eosinophiles. The duration of the attack depends upon the severity and the grade of infection. The mortality ranges from 2% to 30%.

The prophylaxis depends upon the careful examination of the meat, and when pork or sausage is partaken of, it should be thoroughly boiled.

Treatment.—A brisk purge early is advised, and the use of glycerin in large doses to destroy the worm has been recommended by some authorities. Diarrhea early in the course of the infection is favorable. When pains become manifest, they should be relieved by suitable remedies. The patient's strength should be supported. No drug is known that is of value in destroying the worm after it has reached the muscle-tissue.

Filaria.—The female is about 4 cm. in length. The larva, as found in the blood of the living subject, is 0.0075 mm. in breadth and 0.34 mm. in length. The worm has a long, rounded head, with a tongue-like appendage and a short, pointed tail. The parasite is found in the blood and lymph-channels, and occurs particularly in the tropics; it has, however, been occasionally met with in temperate climates.

The parasite may be present a long time in the blood without giving rise to symptoms. In a number of instances, however, it blocks or perforates the capsules or lymphatics, leading to chyluria, hematuria, or hemorrhage in different organs.

According to some recent investigations, it has been shown that the parasite is conveyed by the mosquito. It has been found that one variety of the parasite (filaria nocturna) can be seen in the peripheral blood only at night; another variety (filaria diurna), during the day; while still another variety (filaria perstans) can be found by day as well as by night.

Some forms of elephantiasis are occasionally due to the filaria, and sometimes immense thickening of the tissues of the scrotum occurs. In these cases the parasites are not always found.

A variety of filaria known as *dracunculus medinensis* (guinea-worm disease) occurs in certain parts of Africa and the East Indies. It has extremely rarely been met with in this country.

The worm is usually solitary, is cylindric in form, about 2 mm. in diameter, and from 50 to 80 mm. in length. Only the female is known. The worm gains entrance through the stomach and not through the skin.

Other forms of filaria are known: The filaria loa, which has been found in the conjunctiva; the filaria lentis, occurring in cataract; the filaria labialis; the filaria hominis oris, occurring in the lip and in the mouth; and the filaria bronchialis, which has been found in the trachea and the bronchi.

Other Nematodes.—The **trichocephalus dispar,** or whip-worm, has been found in the large intestine and cecum, the female measuring from 4 to 5 cm., while the male is somewhat shorter. As many as a thousand have been counted in one case. It rarely causes symptoms. The worm is voided in the feces, the eggs being dark brown and lemon-shaped.

Eustrongylus Gigas.—This measures about 30 cm. in length; the female, about 100 cm. It is rarely met with in man. When found in the human subject, it is encountered in the renal region and may destroy the kidney.

Rhabdonema Intestinale.—These are the small worms commonly found in the feces, occurring in the endemic diarrhea of tropical climates. They are often found in connection with the ankylostoma; occasionally they are found in the biliary and pancreatic ducts. When present in large numbers, they give rise to intestinal disturbance with anemia.

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